THE ROLES OF INTRUSIVE VISUAL IMAGERY AND VISUAL IMAGERY ABILITY IN INSOMNIA DISORDER

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ABSTRACT

THE ROLES OF INTRUSIVE VISUAL IMAGERY AND VISUAL IMAGERY ABILITY IN INSOMNIA DISORDER

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A number of theoretical models have been proposed to understand and explain insomnia disorder and its etiology. The present thesis argued that intrusive visual imagery (IVI) in the pre-sleep period may lead to classical conditioning which evokes and perpetuates hyperarousal in chronic insomnia. In Study 1 (N = 651), Pre-sleep Arousal Scale (PSAS) and Intrusive Visual Imagery and Verbal Thoughts Questionnaires (IVIVTQ) were adapted to Turkish. Factor structures of the Turkish forms were tested with exploratory factor analysis and confirmatory factor analysis in split half samples. The validities (convergent, divergent, incremental, discriminant) and reliabilities (internal consistency and test-retest) of the Turkish forms were also assessed in a subsample (N = 556) and a separate third sample (N = 88). The results of Study 1 showed that the Turkish forms of PSAS and IVIVTQ had good validity and reliability features. In Study 2, a path model comprising the variables of intrusive visual imagery (IVI), intrusive verbal thoughts (IVT), visual imagery ability (VIA), pre-sleep arousal (PSA), and insomnia severity (IS) was tested with 168 of 1444 participants who met DSM-5 criteria for Insomnia Disorder. The results showed that IVI, but not IVT, significantly predicted PSA and PSA predicted

IS. Furthermore, the indirect effects of IVI via PSA on IS was significant. Finally, the moderator role of VIA on the relationship between IVI and PSA was not significant. The results of the studies were discussed in the light of existing literature. Clinical implications and suggestions for the future studies were proposed.

Keywords: Insomnia, Intrusive Thoughts, Visual Imagery, Sleep, Fear

ÖZ

UYKUSUZLUK BOZUKLUĞU'NDA İSTENMEYEN GÖRSEL İMGELERİN VE GÖRSEL İMGELEME YETENEĞİNİN ROLÜ

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Uykusuzluk bozukluğunu ve etiyolojisini açıklamak için çok sayıda kuramsal model ortaya atılmıştır. Bu çalışma, uyku öncesinde zihinde oluşan istenmeyen görsel imgelemenin (İGİ) kronik uykusuzlukta aşırı uyarılmayı açığa çıkaran ve onu sürdüren klasik koşullanmaya yol açabileceğini öne sürmektedir. Çalışma 1'de (N = 651), Uyku Öncesi Uyarılma Ölçeği (UÖUÖ) ve İstenmeyen Görsel İmgeleme ve Sözlü Düşünceler Anketileri (İGİSDA) Türkçeye uyarlanmıştır. Türkçe formların faktör yapıları, yarıya bölünmüş örneklemlerde açımlayıcı faktör analizi ve doğrulayıcı faktör analizi ile test edilmiştir. Türkçe formların geçerlilikleri (yakınsak, ıraksak, artımlı, ayırt edici) ve güvenirlikleri (iç tutarlılık ve test-tekrar test) bir alt örneklemde (N = 556) ve üçüncü bağımsız bir örneklemde (N = 88) değerlendirilmiştir. Çalışma 1'in sonuçları, UÖUÖ ve İGİSDA'nın Türkçe formlarının iyi geçerlilik ve güvenilirlik özelliklerine sahip olduğunu göstermiştir. İkinci çalışmada istenmeyen görsel imgeleme (İGİ), istenmeyen sözel düşünceler (İSD), görsel imgeleme yeteneği (GİY), uyku öncesi uyarılma (UÖU) ve uykusuzluk şiddeti (UŞ) değişkenlerinden oluşan bir yol modeli, Uykusuzluk Bozukluğu için DSM-5 kriterlerini karşılayan 1444 katılımcıdan 168'inin verileri ile test edilmiştir.

Sonuçlar, İSD’nin değil ancak İGİ'in UÖU’yı anlamlı olarak yordadığı ve UÖU'nun UŞ'yi yordadığını göstermiştir. Ayrıca, İGİ’nin UÖU üzerinden UŞ’yi anlamlı olarak etkilediği bulunmuştur. Son olarak İGİ ve UÖU arasındaki ilişkide GİY'nin düzenleyici rolü olmadığı açığa çıkmıştır. Mevcut çalışmaların sonuçları literatür ışığında tartışılmıştır. Gelecekti çalışmalar için klinik çıkarımlar ve öneriler belirtilmiştir.

Anahtar Kelimeler: Uykusuzluk, İstenmeyen Düşünceler, Görsel İmgeleme, Uyku, Korku

To My Beloved Mother…

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LIST OF ABBREVIATIONS

AHR	Adjusted Hazard Ratio
AOR	Adjusted Odds Ratio
BAI	Beck Anxiety Inventory
CBT-I	Cognitive-Behavioral Therapy for Insomnia
CFA	Confirmatory Factor Analysis
CR	Conditioned Response
CS	Conditioned Stimulus
DASS	Depression Anxiety Stress Scale
DSM-5	The Diagnostic and Statistical Manual of Mental Disorders-5
EFA	Exploratory Factor Analysis
ES	Effect Size
FSMI	Fear Simulation Model of Insomnia
GRAS	Gender Role Attitudes Scale
ICDS-3	International Classification of Sleep Disorders-3
IE	Indirect Effect
ISI	Insomnia Severity Index
IVI	Intrusive Visual Imagery
IVT	Intrusive Verbal Thoughts
IVIVTQ	Intrusive Visual Imagery and Verbal Thoughts Questionnaires
NREM	Non-Rapid Eye Movement
OR	Odds Ratio
OSIVQ	Object-Spatial Imagery and Verbal Questionnaire
PSA	Pre-sleep Arousal
PSAS	Pre-sleep Arousal Scale
REM	Rapid Eye Movement
RR	Relative Risk
RTSQ	Ruminative Thought Style Questionnaire

UR	Unconditioned Response
US	Unconditioned Stimulus
VIA	Visual Imagery Ability
VMI	Visual Mental Imagery
VP	Visual Perception

CHAPTER 1

INTRODUCTION

"Now I lay me down to sleep
I pray the Lord my soul to keep
If I die before I wake
I pray the Lord my soul to take"

Metallica – Enter Sandman, 1991

An Irish proverb says, "A good laugh and a long sleep are the best cures in the doctor's book." For some people, however, such simple cures are unfortunately missing. The English noun *insomnia* comes from the Latin word *somnus* (sleep) and the prefix *in* (without), which essentially means *sleepless*. It is known that insomnia has been a problem for humans since ancient times. There are many mentions of insomnia and its remedies in Ancient Greek and Egyptian texts (Asaad, 2015; Montiglio, 2015). For instance, Ancient Egyptians recommended using the poppy seed, lavender, and chamomile as cures for insomnia (Asaad, 2015).

Even though insomnia is generally characterized as not being able to sleep, it also includes sleep problems of initiating sleep, maintaining sleep, early morning awakenings, and complaints about sleep duration and quality. Insomnia may be an acute problem or proceed as a chronic complaint. Longitudinal studies revealed that after the development of insomnia, the complaints were maintained for a year for 70% of individuals and up to three years for 50% of them (Morin & Benca, 2012). Previously, researchers have proposed a number of insomnia models to explain the etiology and maintenance of insomnia disorder (Perlis et al., 2016). The purpose of the present study was to test a model emphasizing the roles of intrusive visual

imagery (IVI) and visual imagery ability (VIA) in the formation of primary insomnia disorder.

1.1 Definition of Insomnia Disorder

While it is possible to define insomnia as not being able to sleep or having sleep problems, insomnia disorder is a stand-alone disorder that is characterized by subjective complaints about sleep quality and length (Qazi & Schluederberg, 2012; Winkelman, 2015). Insomniacs suffer from three main sleep problems: (1) inability to fall asleep, (2) inability to maintain sleeping, and (3) early morning awakenings (Reeve & Bailes, 2010). Many insomniacs have difficulty falling asleep. While non-insomniacs typically fall asleep in twenty minutes (Thomas & Anderson, 2013), it takes longer to fall asleep for insomniacs. Also, the sleep of insomniacs is frequently disturbed, and they cannot quickly resume their sleep. Finally, some insomniacs wake up much earlier than their usual time in the morning hours. As a result of these sleep difficulties, insomniacs frequently complain about reduced sleep quality and length and report impairments in daytime functioning such as fatigue, general distress, and low well-being (Hamilton et al., 2007; Morin & Benca, 2012; Winkelman, 2015). Insomnia is regarded as primary if it is not explained by other sleep-related, physical, or mental disorders (Mai & Buysse, 2008). On the other hand, secondary insomnia may be caused by mental disorders, medical conditions, breathing disorders during sleep, and other sleep disorders (Ohayon, 2002).

Insomnia subtypes are identified either by the duration of insomnia or major insomnia symptoms (Krystal, 2005). The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) includes three subtypes of insomnia based on duration, which are acute (less than one month), episodic (at least one month but less than three months), and persistent (more than three months) (American Psychiatric Association, 2013). Similarly, the International Classification of Sleep Disorders-3 divides insomnia into two subtypes short-term insomnia (less than three months) and chronic insomnia (more than three months) (Grima et al., 2019). Investigating the transition from acute insomnia to chronic insomnia, Perlis et al. (2020) asked 1248 good sleepers to

complete sleep diaries and other health measures for a year. The results revealed that 27% of the participants had acute insomnia in the one-year period, 72.4% of those participants recovered, and 1.8% of them developed chronic insomnia.

In terms of symptom-based classification, three subtypes of insomnia have been generally classified as sleep-onset insomnia, sleep-maintaining insomnia, and insomnia with early morning awakening (Reeve & Bailes, 2010). Previous studies also suggested that insomniacs may have multiple overlapping insomnia symptoms, and their complaints may change over time (Krystal, 2005). The results of the 1991 National Sleep Foundation Survey (n = 1000) indicated that 56%, 67%, and 44% of insomniacs had difficulty falling asleep, disturbed sleep, and early morning awakenings, respectively (Ancoli-Israel & Roth, 1999). Hohagen (1994) examined the temporal stability of insomnia subtypes in a sample of 328 patients. It was found that 47% of general insomniacs (who have all insomnia symptoms), 51% of sleep-onset insomniacs, 17% of sleep maintaining insomniacs, and 45% of early morning awakening insomniacs remained in their baseline groups at four months of follow-up. Blanken et al. (2019) conducted a latent class analysis of 2224 participants who completed 34 questionnaires regarding personality and affect traits, sleep, life events, and health history. The results revealed five subtypes of insomnia: highly distressed, moderately distressed but reward sensitive (to pleasurable emotions), moderately distressed and insensitive, slightly distressed with high reactivity (to their environment and life events), and slightly distressed with low reactivity.

Another intriguing type of insomnia is paradoxical insomnia, in which there are discrepancies between subjective complaints of insomnia and the objective findings regarding sleep (Castelnovo et al., 2019; Rezaie et al., 2018). Bonnet and Arand (2010, p. 13) described people with paradoxical insomnia as “a group of patients who complain of insomnia but have perfectly normal sleep as measured by their EEG sleep stages.” People with paradoxical insomnia complain about insufficient sleep and short total sleep time, despite the lack of objective evidence, such as polysomnographic sleep findings. Polysomnographic measures are sets of

physiological measures recorded during sleep, such as brain waves, heart rhythm, muscle tone, body movements, eye movements, and breathing patterns (Rezaie et al., 2018). It has been found that the prevalence of paradoxical insomnia is between 8-66% depending on the definition used (Castelnovo et al., 2019; Rezaie et al., 2018). Although the etiology of paradoxical insomnia has not been fully revealed, recent studies indicated that nighttime micro-awakenings, heightened cortical arousal, and REM sleep macrostructure differences may be causes of paradoxical insomnia (Parrino et al., 2009; Pérusse et al., 2015; Rezaie et al., 2018). Despite the variety of insomnia types based on duration or symptoms, two major tools generally guide diagnostic procedures for insomnia disorder.

1.2 Diagnosis of Insomnia Disorder

The most commonly used tools to diagnose insomnia are DSM-5 and ICDS-3. DSM-5 diagnostic criteria for insomnia disorders can be seen in Table 1. DSM-5 diagnosis for persistent insomnia disorder requires one or more symptoms of difficulty falling asleep, disturbed sleep, and early morning awakenings being present for at least three nights a week and for at least three months. In addition, overall sleep difficulties must lead to significant distress and impairment in several life areas such as social, occupational, and educational areas. Finally, the sleep difficulty must be present even if there is adequate opportunity for sleep, and sleep disturbances must not be explained by any other sleep disorder, physical illness, or mental disorder.

Table 1 The DSM-V Diagnostic Criteria for Insomnia Disorder

1) Dissatisfaction with sleep quantity or quality, associated with one (or more) of the following symptoms:
a. Difficulty initiating sleep (for children, in the absence of a caregiver)
b. Difficulty maintaining sleep (for children, in the absence of a caregiver)
c. Early-morning awakening with inability to return to sleep.
2) Clinically significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning.
3) Occurs at least three nights per week.
4) Present for at least three months.
5) Occurs despite adequate opportunity for sleep.
6) Not better explained by and does not occur exclusively during the course of another sleep-wake disorder.

Table 1 (continued)

7) Not attributable to the physiological effects of a substance.
8) Not better explained by coexisting mental disorders and medical conditions

(Adapted from American Psychiatric Association, 2013, p. 362)

The clinicians are asked to specify comorbidity as either *with non-sleep disorder mental comorbidity, with other medical comorbidity,* or *with other sleep disorder*. It is also required to state whether the disorder is *episodic* (1-3 months), *persistent* (more than three months), or *recurrent* (two or more episodes in a year period). Finally, acute insomnia (symptoms present for less than three months) is defined as *other specified insomnia disorder*.

The criteria for chronic insomnia disorder in the International Classification of Sleep Disorders-3 can be seen in Table 2. The ICDS-3 criteria for chronic insomnia disorder are very similar to the DSM-5 criteria (American Academy of Sleep Medicine, 2014). Alongside three basic insomnia symptoms, major insomnia complaints must also include *resistance to going to bed on appropriate schedule* and *difficulty sleeping without parent or caregiver intervention*. ICDS-3 also asks for consequences of insomnia such as fatigue, daytime sleepiness, reduced energy, etc. The criteria regarding frequency and duration of the symptoms are *at least three nights per week* and *at least three months*, respectively. Similar to DSM-5, sleep difficulty must be present despite adequate opportunity for sleep. Finally, the sleep difficulty must not be explained by another sleep or mental disorder, medical condition, or the effects of a substance (American Academy of Sleep Medicine, 2014). The diagnostic criteria for insomnia in the DSM-5 and ICDS-3 enable researchers to examine the prevalence of insomnia in population-based studies.

Table 2 The diagnostic criteria for chronic insomnia disorder in the ICDS-3

1) Sleep problem (one or more):
a. Difficulty initiating sleep
b. Difficulty maintaining sleep
c. Waking up earlier than desired time
d. Resistance to going to bed on appropriate schedule
e. Difficulty sleeping without parent or caregiver intervention

Table 2 (continued)

2) Consequences (one or more):
a. Fatigue/malaise
b. Attention, concentration, or memory impairment
c. Impaired social, family, occupational, or academic performance
d. Mood disturbance, irritability
e. Daytime sleepiness
f. Behavioral problems (e.g., hyperactivity, impulsivity, aggression)
g. Reduced motivation, energy, initiative
h. Proneness for errors, accidents
i. Concerns about or dissatisfaction with sleep
3) Frequency
Occur at least three nights per week.
5) Adequate opportunity
Occurs despite adequate opportunity and circumstances for sleep.
6) Relationship to another condition
Not better explained by another sleep disorder, a coexisting mental disorder, coexisting medical condition or the physiologic effects of a substance.

(Adapted from American Academy of Sleep Medicine, 2014, pp. 21–22)

1.3 Prevalence of Insomnia

The prevalence rate of insomnia disorder changes depending on the strictness of definitions and diagnostic criteria (Bos & Macedo, 2019; Ohayon, 2002). Before major diagnostic tools included insomnia as a separate diagnosis, there had been controversy in the literature about defining insomnia. Researchers have proposed several insomnia subtypes according to frequency, duration, and etiology. Furthermore, insomnia has been regarded as either a symptom of a comorbid disorder or a separate sleep disorder (Mai & Buysse, 2008). Over the years, primary diagnostic tools (DSM-IV and ICSD-2) began to distinguish secondary insomnia caused by other medical conditions and mental disorders from primary insomnia, which is non-organic and develops independently from any medical and mental disorders (Mai & Buysse, 2008; Reeve & Bailes, 2010). While up to 30% of the general population reports at least one insomnia symptom (Ohayon, 2002), the prevalence studies found that only 5-19.2% of the adult population met all DSM-IV criteria for insomnia disorder in Chinese, Canadian, French, Norwegian, American, Spanish, and South Korean samples (Cao et al., 2017; Ford et al., 2015; Leger et al., 2000; Morin et al., 2011; Ohayon & Hong, 2002; Ohayon & Sagales, 2010; Pallesen et al., 2001). In their epidemiological study with 4758 participants, Benbir et al. (2015) found that

half of the Turkish participants reported at least one insomnia symptom, and 12.2% of them met insomnia criteria adapted from the DSM-IV and ICDS-2. The prevalence rates of insomnia in various national samples indicate that insomnia may bring about substantial personal and societal consequences.

1.4 Consequences of Insomnia

Insomnia brings several personal and societal costs, such as decreased quality of life, decreased daytime functioning, impaired cognitive skills, reduced work performance, increased absenteeism, increased treatment costs for non-insomnia disorders, more frequent visits to health-care institutions, and hospitalization (Wade, 2010). LeBlanc et al. (2007) compared three groups of people who have insomnia syndrome (n = 147), insomnia symptoms (n = 308), and good sleepers (n = 493). The results indicated that individuals with insomnia syndrome reported decreased health-related quality of life and had higher scores on the measures of depression, anxiety, neuroticism, extraversion, arousal predisposition, stress perception, and emotion-oriented coping compared to individuals with insomnia symptoms and good sleepers (LeBlanc et al., 2007). In a cross-sectional survey study with 4067 people from the US, France, and Japan, Léger et al. (2012) revealed that chronic insomniacs had lower health survey scores (Short Form-36) in all dimensions (physical functioning, limitations due to physical health, bodily pain, general health perceptions, vitality, social functioning, limitations due to emotional problems, and mental health) compared to good sleepers. Sofi et al. (2014) examined 13 studies, including 122,501 participants. The results showed that insomnia increased the risk of developing or dying from cardiovascular disease (RR = 1.45, 95% CI [1.29–1.62]). Similar results were also found for hypertension (Javaheri & Redline, 2017). Also, Godet-Cayré et al. (2006) found that insomniacs were significantly more absent from work (5.8 days) compared to good sleepers (2.4 days).

In their meta-analysis study examining the effects of insomnia on daytime cognitive performance, Fortier-Brochu et al. (2012) showed that insomniacs suffer from mild to moderate impairments on tasks testing episodic memory (ES = -.51), problem-

solving (ES = −.42), manipulation in working memory (ES = -.42), and retention in working memory (ES = -.22). On the other hand, there were no significant differences between the performance of insomniacs and normal sleepers in terms of general cognitive functioning, perceptual processes, psychomotor functions, verbal functions, alertness, divided attention, sustained attention, vigilance, procedural memory, cognitive flexibility, and verbal fluency (Fortier-Brochu et al., 2012).

These personal and societal costs can also be expressed in monetary terms. By combining the estimated direct and indirect medical costs of insomnia, such as loss of productivity, insomnia-induced depression, alcohol abuse, and accidents, Stoller (1994) calculated the total annual cost of insomnia in the United States to be within the range of $92.5-107.5 billion, which was equal to $150.36-174.89 billion when the costs are adjusted for inflation in 2016 (Reynolds & Ebben, 2017). In Canada, Daley et al. (2010) estimated the total annual cost of insomnia in the province of Quebec as 6.6 billion Canadian dollars. In addition to the personal and societal costs, insomnia may also be a risk factor for various mental health problems.

1.5 Insomnia and Psychopathology

A great deal of previous research into insomnia has assessed the role of insomnia as a risk factor for psychopathologies. In addition to being a disorder by itself, insomnia is frequently a symptom of other mental disorders such as major depressive disorder or post-traumatic stress disorder (Harvey, 2001; Inman et al., 1990). In their study with 14,915 participants, Ohayon & Roth (2003) found that insomnia precedes mood and anxiety disorders in 41% and 18% of cases, respectively. Thus far, the studies have suggested that insomnia may be a precipitating factor for mental illnesses, and the highest risk is for depression. (Pigeon et al., 2017).

Several lines of evidence consistently indicated that insomnia might precipitate and be causally linked to depression (Staner, 2010). Nearly 80% of major depressive disorder patients have comorbid insomnia, and in half of these cases, insomnia precedes the onset of major depressive disorder (Ohayon & Roth, 2003). Reviewing

eight epidemiological studies, Riemann and Voderholzer (2003) found that having insomnia at baseline was a significant predictor of an increased depression risk at 1-3 years of follow-ups. In their meta-analysis of 21 studies, Baglioni et al. (2011) showed that the overall odds ratio for insomnia to predict depression was 2.60 (95% CI = 1.98-3.42), and it was 2.10 when adjusted for outliers (95% CI = 1.86–2.38). In their prospective population-based studies (the HUNT2 in 1995–1997 and the HUNT3 in 2006–2008), Siversten et al. (2012) found that the odds ratio of developing depression at HUNT3 for participants without depression in HUNT2 but having insomnia in HUNT2 and HUNT3 was 6.21 (95% CI = 4.45-8.68), and it was 5.67 when adjusted for outliers (95% CI = 3.52-9.12). On the other hand, the odds ratio of developing insomnia at HUNT3 for participants without insomnia in HUNT2, but having depression in HUNT2 and HUNT3 was 6.7 (95% CI = 5.46-8.26), and it was 4.96 when adjusted for outliers (95% CI = 3.65-6.75).

At this point, how insomnia can lead to depression must be addressed. Previous research has established that sleep deprivation has a short-term antidepressant effect on depressive symptoms, although its mechanism of action is unclear (Hemmeter et al., 2010; Hines et al., 2013; Wu & Bunney, 1990). Staner (2010) suggested that while acute total sleep deprivation (complete elimination of sleep for a period of time) or acute partial sleep deprivation (reducing amount of total sleep time) may have antidepressive effects, chronic sleep deprivation may have a direct causal role in the formation of depressive symptoms. Pigeon et al. (2017) proposed that stress hormone secretion and high levels of inflammatory cytokines triggered by insomnia may lead to depression. Furthermore, the state of despair in the nighttime and insomnia-induced daytime impairments in coping skills may complicate the handling of interpersonal and social problems. Eventually, people may activate their depressive schemas or develop learned helplessness, and these paths may indirectly lead to depression (Pigeon et al., 2017; Staner, 2010).

In addition to depression, insomnia has also been linked to other psychopathologies. Pigeon et al. (2017) reviewed 16 studies about insomnia and psychopathologies. The

results yielded that insomnia was a significant risk factor for having an anxiety disorder over a six-year period (AHR= 2.38, 95% CI = 2.21–2.57), a bipolar disorder over a ten-year period (AOR = 1.41; 95% CI = 1.11–1.79), a substance abuse disorder over a six-year period (AHR = 4.12; [3.45–4.93], and committing suicide over a ten-year period after controlling for depressive symptoms (AOR = 1.30; 95% CI = [1.04 – 1.63]). Similar results were reported by Hertenstein et al.'s meta-analysis (2019). It was found that insomnia was a significant predictor for the onset of depression (OR = 2.83, 95% CI = 1.55–5.17), anxiety (OR = 3.23, 95% CI = 1.52–6.85), alcohol abuse (OR = 1.35, 95% CI = 1.08–1.67), and psychosis (OR = 1.28, 95% CI = 1.03–1.59) (Hertenstein et al., 2019). The current evidence suggests that insomnia is a stand-alone psychiatric disorder and a significant risk factor for various mental health problems. Therefore, it is also important to understand the treatment methods for insomnia.

1.6 Treatment of Insomnia Disorder

In principle, there are two types of recommended treatments for insomnia, which are pharmacotherapy and psychotherapy (Qazi & Schluederberg, 2012; Reeve & Bailes, 2010). Independent of the treatment modality, treatment of chronic insomnia generally aims to satisfy two primary goals: (1) improving the quality and quantity of sleep and (2) ameliorating daytime impairment (Lie et al., 2015). Before selecting appropriate treatment, it is very crucial to comprehend the socioeconomic background of the patient (such as work schedules or shift work) or daily habits like caffeine consumption (Qazi & Schluederberg, 2012). It is also recommended that short-term pharmacological therapies should be supported by behavioral or cognitive psychotherapies when possible (Schutte-Rodin Sharon et al., 2008).

1.6.1 Pharmacological Treatments

The selection of pharmacological agents for the treatment of insomnia requires assessments regarding "(1) symptom pattern; (2) treatment goals; (3) past treatment responses; (4) patient preference; (5) cost; (6) availability of other treatments; (7) comorbid conditions; (8) contraindications; (9) concurrent medication interactions;

and (10) side effects" (Schutte-Rodin Sharon et al., 2008, p. 498). The pharmacological agents utilized in treating insomnia are benzodiazepine receptor agonists, melatonin agonist (ramelteon), tricyclic antidepressant (doxepin), barbiturates, orexin receptor antagonist (suvorexant), antidepressants, atypical antipsychotics, and over-the-counter medications (antihistamines and melatonin) (Lie et al., 2015). The American Academy of Sleep Medicine approved a general sequence for medication trials as follows: (1) short-intermediate acting benzodiazepine receptor agonists (BzRA) or ramelteon; (2) if the initial agent fails, alternate short-intermediate acting BzRAs or ramelteon; (3) sedating antidepressants, especially for insomnia comorbid with depression or anxiety; (4) combined BzRA or ramelteon and sedating antidepressant; (5) other sedating agents like anti-epilepsy medications (gabapentin, tiagabine) and atypical antipsychotics (Schutte-Rodin Sharon et al., 2008). It is also recommended that pharmacological treatment should be assisted by informing patients about treatment goals and expectations, possible side effects and drug interactions, psychological treatments (cognitive-behavioral therapy for insomnia), and a regular assessment of treatment effectiveness (Schutte-Rodin Sharon et al., 2008).

In their meta-analysis of 105 randomized controlled trials, Buscemi et al. (2007) found significant reductions in polysomnographic and sleep-diary sleep-onset latencies for benzodiazepines, non-benzodiazepines, and antidepressants. Huedo-Medina et al. (2012) conducted a meta-analysis of 13 studies (n = 4378) assessing the effects of non-benzodiazepine hypnotics such as eszopiclone, zaleplon, and zolpidem. It was found that there were small to medium effect sizes for polysomnographic (ES = -.36, CI 95% [−.57, −.16]) and subjective sleep latency (ES = -.33, CI 95% [−.62, −.04]), yielding weighted mean raw differences of 22 minutes for drugs (CI 95% [-33, -11]) compared to placebo.

1.6.2 Psychological Therapies

First-line psychotherapy for insomnia is cognitive-behavioral therapy for insomnia (CBT-I). Guided by cognitive and behavioral theories of insomnia, CBT-I aims to (1)

modify dysregulation of sleep drive by stimulus control and sleep restriction; (2) alleviate sleep-related anxiety through cognitive restructuring; and (3) replace sleep-interfering behaviors with functional alternatives (Mitchell et al., 2012). CBT-I lasts 4 to 8 sessions on average and includes components such as sleep restriction (restriction of time spent on the bed to an optimal minimum), stimulus control (banning non-sleep-related activities on the bed during bedtime), cognitive restructuring (changing maladaptive sleep-related thoughts), sleep hygiene education (optimizing sleep environment and avoiding activities such as coffee consumption in the evening, eating and exercising before sleep), and relaxation training (Mitchell et al., 2012; Siebern et al., 2012).

Although recent research has shown that CBT-I is effective for treating insomnia (Harvey et al., 2014; Mitchell et al., 2012; van der Zweerde et al., 2019), the findings indicate two weak points regarding the effect of CBT-I, which are the duration of the effect and response-remission rates. In their meta-analysis of 30 randomized controlled trials, van der Zweerde et al. (2019) found that the effects of CBT-I decline over time. The results yielded low to moderate effect sizes (Hedges' g) across three-time intervals for insomnia severity (3 months follow-up, 0.64; 6 months follow-up, 0.40; 12 months follow-up, 0.25), sleep-onset latency (3 months, 0.38; 6 months, 0.29; 12 months, 0.40), and sleep efficiency (3 months follow-up, 0.51; 6 months follow-up, 0.32; 12 months follow-up, 0.35). Harvey et al. (2014) also compared 188 chronic insomnia patients who were randomly assigned to behavior therapy (BT, n = 63), cognitive therapy (CT, n = 65), or cognitive-behavioral therapy (CBT, n = 60) in terms of response and remission rates. The results indicated that 67.25% of CBT (6 months, 67.55%), 67.35% of BT (6 months, 44.44%), and 42.2% of CT (6 months, 62.59%) subjects responded to treatments. Moreover, remission rates at post-treatment were 57.29% for CBT (6 months, 55.82%), 39.4% for BT (6 months, 36.45%), and 30.84% for CT (6 months, 51.62%), meaning that hardly half of the patient benefited from CBT-I. In summary, the best available psychotherapy for insomnia provides relief for only half of the insomniacs, and the effect of the treatment declines significantly over a year. In order to develop models explaining

insomnia and effective treatment methods for insomnia disorder, sociodemographic, physiological, and psychological factors that distinguish insomniacs from good sleepers may provide important insights.

1.7 Sociodemographic, Physiological, and Psychological Correlates of Insomnia

1.7.1 Sociodemographic Correlates

Previous studies have shown that gender, age, marital status, and socioeconomic status were associated with insomnia disorder. Compared to males, females report more insomnia symptoms and are more likely to be diagnosed with insomnia disorder (Benbir et al., 2015; Ford et al., 2015; Käppler & Hohagen, 2003; Leger et al., 2000; Morin & Benca, 2012; Ohayon, 2002; Ohayon & Hong, 2002; Pallesen et al., 2001; Sivertsen et al., 2009). It has been asserted that predisposition to anxiety, menstrual symptoms, differences in sex hormones, and menopause may increase the risk of insomnia for females (Soares, 2005). Furthermore, studies have constantly associated advanced age with insomnia (Ford et al., 2015; Klink et al., 1992; Ohayon & Hong, 2002; Pallesen et al., 2001; Sivertsen et al., 2009). Proposed reasons for this relationship are age-specific neurological changes, deteriorated health, medication use, low level of outdoor light exposure, and decreased social contact (Ancoli-Israel, 2005; Gooneratne & Vitiello, 2014; Pallesen et al., 2001). However, one large study with 13057 participants yielded that age was not a significant predictor of insomnia symptoms after controlling activity status and social life satisfaction (Ohayon et al., 2001). In a meta-analysis study with 115,988 participants, Cao et al. (2017) found a reverse pattern in a Chinese sample, showing that younger participants had a higher pooled insomnia prevalence than older participants. In terms of marital status, singles were less likely to report insomnia compared to married or divorced individuals (Benbir et al., 2015; Kawata et al., 2020; Leger et al., 2000). Lower individual and household education were also found to be significant predictors of insomnia even after controlling for age, gender, and ethnicity (Gellis et al., 2005; Sivertsen et al., 2009).

1.7.2 Physiological Correlates

Researchers have shown that insomniacs and good sleepers differ in terms of a number of physiological measures such as body temperature, cortisol levels, electrodermal activity, heart rate and variability, and metabolic rate, suggesting that hyperarousal may play an important role in insomnia. Reviewing the literature regarding body temperature rhythms in insomniacs, Lack et al. (2008) proposed that insomnia subtypes may be associated with delayed or advanced sleep phase disorders in which circadian rhythms are distorted. Normally, people fall asleep 5-6 hours before their core body temperature (CBT) reaches its minimum level and wake 1-3 hours after $CBT_{minimum}$ (Lack et al., 2008). Morris et al. (1990) compared 13 sleep-onset insomniacs and nine normal sleepers in terms of body temperature rhythms. The results yielded that insomniacs' temperature rhythms were delayed by 2.5 hours compared to normal sleepers. Moreover, in their study with early morning awakening insomniacs, Lack et al. (1996) found that insomniacs' ($n = 10$) $CBT_{minimum}$ occurred 4 hours earlier than normal sleepers ($n = 8$). The results pertaining to an earlier than normal $CBT_{minimum}$ were also supported by another study in which sleep diaries and actigraphy were used (Lack et al., 2005). These findings indicate that insomniacs try to fall asleep either earlier or later than their circadian and body temperature rhythms allow them to sleep.

A meta-analysis of 20 studies conducted by Dressle et al. (2022) showed that patients with insomnia have higher cortisol levels (standardized mean differences: 0.50, 95% CI: [0.21–0.80]) both during the day and at night compared to good sleepers, supporting the hyperarousal-insomnia relationship. In their study, Waters et al. (1993) compared 14 sleep-onset insomniacs, 13 sleep-maintenance and mixed insomniacs, and 13 good sleepers. The researchers exposed the participants to orienting response habituation (gradual decrease of central nervous system activity as a result of repeated stimulus presentation) and emotional stress elicitation interventions during which the participants' several electrophysiological responses were measured. It was found that increased electrodermal activity was correlated with poor sleep ratings. Similar results were also found by Broman and Hetta (1994a),

showing that insomnia patients had higher electrodermal activity during the habituation paradigm. Studies undertaken so far provided conflicting evidence concerning heart rate and heart rate variability differences in insomnia patients. A critical review of 22 studies investigating heart rate and heart rate variability in insomnia disorder showed that the majority of the studies found non-significant differences between insomnia patients and good sleepers (Dodds et al., 2017). Dodds et al. (2017) concluded that heart rate variability impairment in insomnia did not have strong empirical evidence. Reviewing four studies examining the metabolic rate in insomniacs and good sleepers, Chapman et al. (2018) found that insomniacs have a higher 24-hour metabolic rate compared to good sleepers. Overall evidence indicated that insomnia patients had elevated autonomic measures characterizing hyperarousal.

1.7.3 Psychological Correlates

Various studies also investigated the differences between insomniacs and good sleepers in terms of personality characteristics. Two previous studies found that insomniacs also had significantly higher harm avoidance, a temperament factor characterized by excessive worry, fear of uncertainty, shyness, being easily fatigued, and lower self-directedness scores than good sleepers (Bravo-Ortiz et al., 2013; de Saint Hilaire et al., 2005). De Saint Hilaire et al. (2005, p. 189) described harm avoidance (HA) as the following: “HA is related to brain systems involving behavioral inhibition. HA is the tendency to respond intensely to aversive stimuli, thereby facilitating learning to inhibit behavior in order to avoid punishment and frustrative omission of respected rewards.”.

In their study with 259 insomnia patients and 137 controls, Lundh and Broman (2006) revealed that insomniacs had significantly higher alexithymia scores than the controls. However, the significant difference disappeared after controlling for trait anxiety. In terms of subscales, insomniacs reported significantly higher externally oriented thinking (a thinking style focusing on external events while excluding inner experience) scores than the controls. Moreover, Gurtman et al. (2014) found that people with self-reported insomnia had significantly higher neuroticism and openness

to experience scores but lower conscientiousness scores compared to good sleepers. In their network analysis study with 2089 participants, Dekker et al. (2017) revealed that there were significant correlations between insomnia severity and neuroticism, agreeableness, and openness to experience (r = .38, r = .12, and r = .10, respectively). Akram et al. (2018) compared 200 Type D personality participants, characterized by social inhibition and negative affectivity, and 192 non-Type D personality participants in terms of insomnia severity. It was found that individuals with the Type D personality trait reported significantly more symptoms of insomnia than individuals with Non-Type D personality. There were also significant correlations between insomnia severity and negative affectivity (r = .42) and social inhibition (r = .25). Akram et al. (2018) examined 475 individuals in regards to dark triad personality traits, which include Machiavellianism (being manipulative, callous, and strategic), psychopathy, and narcissism. The results indicated that there were small significant correlations between insomnia severity and Machiavellianism (r = .14) and psychopathy (r = .15). Moreover, psychopathy but not Machiavellianism was a significant predictor of insomnia symptoms (Akram et al., 2018). In general, revealing sociodemographic, physiological, and psychological correlates of insomnia leads researchers to develop models that determine predisposing factors of insomnia, how it begins and how it is perpetuated.

1.8 Models of Insomnia

One of the distinct features of insomnia disorder is that there has been an ongoing effort to understand it from different theoretical perspectives. Over the past five decades, researchers have proposed a number of models to explain insomnia disorder (Perlis et al., 2016). While some of the models stressed the significance of psychological and behavioral factors such as conditioning or cognitive appraisals, others underlined the importance of physiological or neurological changes concomitant to insomnia. There are also eclectic models that collate both psychological and physiological factors and strive to present a more holistic view of insomnia. The models developed to elucidate insomnia have significance because treatment methods for insomnia disorder rely on these models considerably.

1.8.1 Stimulus Control Model (1972)

The stimulus control model was proposed by Bootzin (1972) to explain how operant conditioning plays a role in the formation of insomnia. When a stimulus is constantly paired with a single behavior, it becomes more likely that the stimulus will conceive only the associated response (*Discriminative Stimulus – APA Dictionary of Psychology*, n.d.; Perlis et al., 2016). In this model, falling asleep is regarded as an instrumental act, and sleep has the role of being a reinforcer. As a result, sleep-related stimuli, such as bedroom, bed, and bedtime, function as discriminative stimuli for the occurrence of the reinforcement, sleep (Bootzin, 1972). Difficulty in falling asleep may occur if sleep-related cues are associated with non-sleep cues (stimulus dyscontrol) such as eating in bed, working in bed, or watching TV in bed; these behaviors promote wakefulness instead of sleep. Previous research to test stimulus control theory has established that stimulus control with and without sleep restriction produced promising outcomes in terms of sleep latency, the number of awakenings, wake time after sleep onset, total sleep time, sleep quality ratings, and sleep efficiency (Murtagh & Greenwood, 1995; Riedel et al., 1998; Smith et al., 2002). Despite the effectiveness of stimulus control as a therapy for insomnia, Perlis et al. (2016) mentioned two shortcomings of the model. The first one is that there is not enough evidence to show that stimulus dyscontrol is related to the predisposition to, the formation of, and maintenance of insomnia. Secondly, the model particularly emphasizes the role of operant conditioning, meaning the role of classical conditioning in insomnia may be overlooked by the model.

1.8.2 The Spielman Model (3P Model) (1987)

The Spielman Model also called the Three-Factor Model or 3P model, conceptualized insomnia as the interaction of predisposing, precipitating, and perpetuating factors (Spielman et al., 1987). Predisposing factors form the background characteristics that increase the likelihood of having insomnia. These factors include genetic vulnerabilities, trait hyperarousal, trait worry and rumination, or having forced sleep schedules. Precipitating factors include acute problems leading to acute insomnia, such as stressful life events, physical illness, or mental disorders (Perlis et al., 2016).

Finally, perpetuating factors refer to the non-sleep-related behaviors developed as responses to sleep problems but, unfortunately, exacerbate sleep difficulties and turn acute insomnia into chronic insomnia. Perpetuating factors comprise unpredictability of sleep time, staying in bed while awake, increased caffeine consumption, going to bed early, getting out of bed late, and napping (Perlis et al., 2016; Spielman et al., 1987). For example, people may try to compensate for daytime impairments by napping and, as a result, further disturb their circadian rhythm. Later, Spielman et al. (2011) updated the model by adding classical conditioning, in which sleep-related stimuli are associated with wakefulness as a perpetuating factor of insomnia.

Focusing particularly on perpetuating factors, the Spielman Model recommends sleep restriction as a therapy for insomnia. Sleep restriction is generally one of the components of CBT-I. In their meta-analysis study, Miller et al. (2014) found that sleep restriction therapy by itself yielded improvements in sleep latency (ES = .64), wake time after sleep onset (ES = 1.36), sleep efficiency (ES = 1.50), total sleep time (ES = .30), total time in bed (ES = 1.26), and sleep quality ratings (ES = .30). Perlis et al. (2016) asserted that existing literature lacks natural history studies that show whether sleep extension, as proposed by the model, is the main factor transforming acute insomnia to chronic insomnia. Thus, further research is needed to confirm the main tenets of the model empirically.

1.8.3 Microanalytical Model (1993)

The Microanalytical model was proposed by Morin (1993). This model comprises four factors, which are arousal, dysfunctional cognitions, maladaptive habits, and consequences, reciprocally affecting each other. A factor can affect the other three factors and can also be affected by them. Arousal could be cognitive, emotional, or physiological and is produced by life factors. Dysfunctional cognitions include worrying, rumination, beliefs, or expectations regarding sleep disturbances. Maladaptive habits refer to compensatory behaviors, such as excessive time spent in bed or napping, to prevent the undesired effects of sleep loss. Finally, the consequences of sleep loss include psychological or social distress, such as daytime

impairment, fatigue, and mood regulation problems. Sleep loss also has negative impact on arousal, dysfunctional cognitions, and maladaptive habits. The model postulates that the interplay between the four factors leads to insomnia. Perlis (2016) noted that while the model successfully sheds light on the self-perpetuating nature of insomnia, it does not explain how insomnia occurs in the first place.

1.8.4 Neurocognitive Model (1997)

Perlis et al. (1997) developed a neurocognitive model to explain the role of the 3P plus classical condition in the formation and maintenance of insomnia. The model conceptualized hyperarousal in three forms, cortical arousal, cognitive arousal, and somatic arousal. While cortical arousal refers to heightened EEG activity (Puzino, Frye, et al., 2019), cognitive arousal includes excessive negatively toned mental activities (Nicassio et al., 1985). Perlis et al. (1997, p. 182) distinguished cortical arousal from cognitive arousal by stating that "…cortical arousal is a form of somatic arousal to the extent that it is a measure of brain, as opposed to mental activity. However, it is also the case that cortical arousal is an analogue of 'cognitive arousal'". Finally, somatic arousal is bodily symptoms such as breathing difficulty, somatic tension, and increased heart rate (Puzino, Amatrudo, et al., 2019).

Among these arousal types, cortical arousal is regarded as central to the etiology and pathophysiology of insomnia. The model posited that cortical arousal during the pre-sleep period, characterized by enhanced sensory processing, information processing, and increased long-term memory function, occurs as a result of classical conditioning. During classical conditioning, sleep-related stimuli such as bedroom, bed, and bedtime elicit cortical hyperarousal, which in turn prevent sleep initiation. According to the model, insomnia is not initiated or maintained by cognitive and somatic hyperarousal but by heightened sensory and information processing triggered by sleep-related stimuli at sleep-onset and during sleep. The model also suggested that insomniacs' subjective perception of their sleep, which is characterized by overestimation of their wakefulness time and underestimation of their sleep time, may be explained by heightened cortical activity during sleep. The model has been

supported by several lines of evidence regarding the difference between insomniacs and good sleepers in terms of cortical or central nervous system arousal (Devoto et al., 2005; Fernandez-Mendoza et al., 2016; Spiegelhalder et al., 2013).

However, Perlis et al. (2016) listed several limitations of the model. First, it does not properly explain the transformation of good sleep into acute insomnia. Second, the model does not include the effects of sleep homeostasis and circadian rhythmicity on sleep. Third, it does not explicitly state which brain regions and circuits are activated around sleep-onset and during NREM sleep. Fourth, it may also neglect the importance of the subcortical regions involved in insomnia and neurological mechanisms underlying the hybrid state of insomnia that is characterized by brain activities related to sleep and wakefulness.

1.8.5 High-Risk Model of Threat Perception (1998)

The High-Risk Model of Threat Perception was theorized by Perlstrom and Wickramasekera (1998) to explain insomnia in terms of somatization and threat perception. The model tries to identify psychosocial and psychophysiological risk factors regarding threat perception and symptoms of insomnia (Wickramasekera, 1993; Wickramasekera, 1995). According to the model, one of the risk factors is hypnotic ability. People who have high and low hypnotic ability may be more prone to developing insomnia (Perlstrom & Wickramasekera, 1998). People with high hypnotic ability are at risk because they are hypersensitive and hyperreactive to the perception of threat. On the other hand, people with low hypnotic ability are also at risk due to their hyposensitivity to emotional threats and parasympathetic dysregulation (inability to restore and calm body functions) during chronic threats (Perlstrom & Wickramasekera, 1998). Another risk factor for insomnia is high overt and covert negative affectivity, which affects the hypothalamic-pituitary-adrenal axis and immune function via chronic negative bias in perception and memory (Perlstrom & Wickramasekera, 1998). These hypotheses were tested in a small sample study (n = 17) by Perlstrom and Wickramasekera (1998). The results showed that there was a

bimodal distribution of high and low hypnotic ability in insomnia patients and insomnia patients had higher negative affectivity scores than the controls.

1.8.6 Sleep Interfering-Interpreting Process Model (2000)

The Sleep Interfering-Interpreting Process Model was developed by Lundh and Broman (2000). The model asserts that there are two psychological processes, sleep-interfering and sleep-interpreting processes, underlying sleep, and insomnia. The sleep-interfering process includes physiological, emotional, and cognitive arousal and the factors influencing the level of arousal such as arousability, frequency and intensity of arousing events, individuals' patterns of emotional regulation, personal stimulus-arousal associations, behavioral and cognitive coping strategies, and interpersonal relations (Lundh & Broman, 2000; Perlis et al., 2016). The sleep-interpreting process includes appraisals of sleep and daytime functioning and the factors influencing these, such as attributions regarding poor sleep and daytime functioning, the level of perfectionism, beliefs about sleep needs, and consequences of poor sleep. According to the model, insomnia occurs as a result of sleep-interfering and interpreting processes through arousal and appraisals. Sleep-interfering processes impede the initiation and maintenance of sleep. Then, sleep-interpreting processes lead individuals to interpret or report their sleep problems as insomnia. For example, people with high arousability may be more likely to be aroused when thinking about interpersonal conflicts during the pre-sleep period. If they are also perfectionists who believe that eight hours of sleep is essential for health, they may be more likely to interpret seven hours of sleep as a serious sleep problem. Perlis et al. (2016) asserted that the model does not clearly explain how acute insomnia becomes chronic insomnia.

1.8.7 Psychobiologic Inhibition Model (2002)

Espie (2002) proposed the Psychobiologic Inhibition Model to explain how inhibition of dearousal processes interferes with normal sleep. While insomnia is generally regarded as a disorder of hyperarousal, the model shifts the emphasis from hyperarousal to inhibition of dearousal. Dearousal processes include sleep-stimulus

control, physiological de-arousal, cognitive dearousal, affect regulation, and daytime facilitation of night-time sleep (Espie, 2002). Sleep-stimulus control refers to associating sleep with sleep-related stimuli (e.g., bedroom, bed, and bedtime), keeping sleep-wake sensitivity/specificity by not lying awake in bed unnecessarily or sleeping during the day, and having regular sleep habits regarding the timing of sleep. Physiological de-arousal is composed of sleep system engagement (e.g., not being tired before sleep or trying too hard to sleep), wake system disengagement (e.g., active thinking and problem-solving or hypervigilance during bedtime), and good sleep hygiene (e.g., not consuming stimulants, not exercising late in the evening or being in a too hot or cold bedroom). Cognitive dearousal is characterized by minimal cognitive drive (e.g., not rehearsing and problem solving in bed, not having a racing mind at sleep-onset or not being preoccupied with sleep and sleeplessness) and accurate sleep-wake attribution (e.g., not having dysfunctional beliefs and attitudes about sleep and consequences of inefficient sleep). Affect regulation is maintained by minimal affect (e.g., no worrying, negative affects, or excitement at sleep-onset) and minimal effort to sleep (e.g., not trying to control sleep and overcome insomnia or monitoring sleepiness states). Finally daytime facilitation of night sleep requires accurate wake-sleep attributions (e.g., not attributing all impaired mood, attention and performance deficiencies to quality of sleep, not blaming insomnia for individual problems, not regarding oneself as insomniac) and effective coping skills in dealing with emotions.

According to the Psychobiologic Inhibition Model, normal sleep is initiated and maintained by the processes of automaticity (involuntary sleep initiation and maintenance) and plasticity (the ability of the system to adapt to external circumstances) (Espie, 2002; Perlis et al., 2016). Accordingly, acute insomnia occurs as one or more of the dearousal processes are inhibited by cognitive, affective, or behavioral processes such as poor stimulus control or affect-laden thinking (Espie, 2002). Normally, acute insomnia may disappear as the stressors resolve, but it may also turn into chronic insomnia. Chronic insomnia may arise if the person shifts attention from a life stressor to insomnia symptoms, tries to initiate sleep

intentionally, and engenders sleep effort and subsequent frustration, which trigger the inhibition of dearousal processes (Perlis et al., 2016). As a result of all of these processes, the automaticity and plasticity of normal sleep are disturbed. The presence of attentional bias regarding insomnia symptoms in the model has been supported by several studies, but its role in the development and maintenance of insomnia requires further investigation (Harris et al., 2015; MacMahon et al., 2006; Marchetti et al., 2006).

1.8.8 Cognitive Model (2002)

The Cognitive Model was developed by Harvey (2002) as an extension of the cognitive theory of anxiety disorders. Cognitive arousal, including rumination and worry, undermines normal sleep and increases the likelihood of developing chronic insomnia. The model suggests that insomnia becomes chronic when one starts to worry about sleep and has a distorted perception of daytime deficits caused by insomnia (Perlis et al., 2016). According to Harvey (2002), when people engage in excessive sleep-related worry about sleep timing, length, or quality or the negative consequences of sleeplessness, it evokes cognitive arousal and distress. As people become more susceptible to sleep-related threats, they begin to attend to and monitor sleep-related threats (Perlis et al., 2016). The sleep-related threats may include body sensations for signs inconsistent with falling asleep, signs of not falling asleep indicated by hearing noises coming from the environment (e.g., noises coming from outside or pets), sleep latency, and impairments in daily functioning (Harvey, 2002). The selective attention and monitoring of sleep-related threats cause the individual to develop a distorted perception of deficits, which further increases excessive sleep-related cognitive activities. The entire process is also exacerbated by dysfunctional beliefs about sleep and safety behaviors such as taking a nap or suppressing sleep-related worry at bedtime (Harvey, 2002). The predictions of the model regarding the role of worry, selective attention, and safety behaviors in insomnia have been supported by several lines of research (Gross & Borkovec, 1982; Haynes et al., 1981; Hiller et al., 2015; Lancee et al., 2017; Woodley & Smith, 2006).

1.8.9 Neurobiological Model (2011)

Buysse et al. (2011) developed the Neurobiological Model to show how persistent activity in wake-promoting neural structures during non-rapid eye movement (NREM) sleep may lead to insomnia symptoms. The model is based on evidence from animal studies and the difference between insomniacs and good sleepers in terms of regional neural activity and metabolism. The studies yielded that NREM sleep of insomniacs is characterized by wake-like neural activity in the prefrontal cortex, paralimbic cortex, thalamus-parietal cortices, and hypothalamic-brainstem (Buysse et al., 2011; Perlis et al., 2016). In other words, this model describes insomnia as a hybrid sleep state comprising brain activities related to both sleep and wakefulness. Perlis et al. (2016) asserted that the model does not give an etiological explanation of how acute insomnia turns into chronic insomnia.

1.8.10 Limitations of The Existing Insomnia Models

In their extensive review of insomnia models, Perlis et al. (2016) listed six aspects of insomnia that have not been adequately explained by most of the existing insomnia models. These are the role of classical conditioning, the effects of normal sleep-wake regulation on sleep problems, differentiating acute from chronic insomnia, gender and age differences regarding chronic insomnia, types and subtypes of insomnia, and the role of elevated arousal or hyperarousal.

Perlis et al. (2016) argued that the role of classical conditioning has been underestimated by most of insomnia models. Two findings supported this proposition. The first one is that CBT-I leads to about 50% symptom reduction in the acute treatment phase (Smith et al., 2002; van der Zweerde et al., 2019), which is below the expected rate. If instrumental conditioning is the only underlying mechanism causing chronic insomnia, this rate should have been higher, because CBT-I specifically targets instrumentally learned behaviors that cause insomnia. The second one is that insomniacs treated with CBT-I may have improvements even after 12 months (Harvey et al., 2014; Mitchell et al., 2012; van der Zweerde et al., 2019). These findings suggested that the additional improvement beyond the acute treatment

phase might have been provided by classical conditioning as successful treatment with short-term CBT-I may condition sleep-related cues with sleep in the long term, although CBT-I does not explicitly target classical conditioning underlying insomnia (Perlis et al., 2016).

In addition to the role of classical conditioning, most models do not include an adequate explanation of normal sleep-wake regulation, including sleep homeostasis and circadian rhythmicity. Disruptions or shifts in these systems may influence predisposing, precipitating, and perpetuating factors leading to insomnia (Perlis et al., 2016). Also, although most models do not include an explanation for the transition from acute insomnia to chronic insomnia, some models include such propositions. Yet, they mostly neglect the etiology and nature of acute insomnia. Implicitly, the models assume that chronic insomnia is an extended version of acute insomnia, despite the lack of findings showing that the same clinical and physiological factors underline both types of insomnia (Perlis et al., 2016; Vargas et al., 2020). Moreover, most models do not explain why women and older adults are more likely to report insomnia. For example, it is wondered why women have a higher prevalence of chronic insomnia, although there are no gender differences in terms of acute insomnia (Perlis et al., 2016). The models generally do not distinguish different subtypes of insomnia, such as sleep-onset insomnia, middle-of-the-night insomnia, early-morning awakenings insomnia, or types of insomnia, such as idiopathic, psychophysiological, or paradoxical insomnia (Perlis et al., 2016). Finally, most models link hyperarousal to acute and chronic insomnia. Yet, there have been no clear findings showing that the relationship between hyperarousal and acute insomnia, as well as hyperarousal and chronic insomnia, are physiologically or psychologically the same (Perlis et al., 2016; Vargas et al., 2020).

Even though a considerable amount of literature has been published on insomnia, insomnia models are still far from presenting a coherent and complete description of how insomnia is precipitated and perpetuated. The present study will strive to explain how classical fear conditioning underlying primary insomnia can be formed by IVI

in the pre-sleep period. The present study argues that IVI simulates fear triggering aversive events, which are modern equivalents of evolutionary threats. At this point, it may be beneficial to also consider a novel perspective that understands sleep in its evolutionary context. Sleep is one of the common activities that we share with other non-human animals, and it has a long evolutionary history.

1.9 Sleep and Insomnia: Evolutionary Perspectives

The evolutionary perspective may provide us with valuable insights deepening our comprehension of sleep and insomnia. Despite the abundance of hypotheses regarding why we sleep, the primary cause of sleep remains unresolved (Lee Kavanau, 2005; Nunn et al., 2016). The current evidence indicates that sleep repairs and clears the neuronal system (Bellesi et al., 2016; Mourrain & Wang, 2019; Reimund, 1994), consolidates learning and long-term memory (Cellini, 2017; Diekelmann & Born, 2010; Klinzing et al., 2019), regulates emotions (Palmer & Alfano, 2017; Sterpenich et al., 2020; Vandekerckhove & Wang, 2017; Wassing et al., 2016), repairs the body (Everson et al., 2014; Van Cauter et al., 2004), modulates immune functions (Bryant et al., 2004; Imeri & Opp, 2009; Lange et al., 2010), and even minimizes the risk of predation (Lima et al., 2005; Lima & Rattenborg, 2007).

While most primate species are arboreal, it was great apes that started building sleep platforms or nests to sleep on trees (Covert, 1995; Duda & Zrzavý, 2013). It appears that building sleep platforms provided the great apes with less arousability and sleep fragmentation (Samson & Shumaker, 2013) and perhaps a higher cognitive capacity (Nunn et al., 2016). Going a step further, humans began to sleep on the ground (terrestrial sleep), which enabled them to have longer and higher quality sleep (Coolidge & Wynn, 2018). Despite the advantage of terrestrial sleep in regards to sleep quality, sleeping on the ground increases the risk of predation. It is believed that humans overcame this problem by utilizing fire and sleeping around it, which can keep predators and insects away (Nunn et al., 2016; Worthman & Melby, 2002).

The sleep of post-industrial people differs significantly from the sleep of hunter-gatherers and other primates in many aspects, such as sleep group size, sleeping platforms, environmental lighting, sleep schedules, and so on (Nunn et al., 2016). According to the schedules determined by the day-night cycle, the hunter-gatherers were sleeping around fires on foliages with groups of approximately 26 people (Worthman & Melby, 2002). Today, people sleep on comfortable beds with a mate or alone, go to sleep and wake up on artificially set schedules. The sleep environment is highly protected from the ancestral threats of predators or social hostility. One of the most significant differences is the lighting of the environment, which used to be controlled by the day-night cycle. Many modern-day people have access to artificial lights, enabling them to function in absence of daylight (Samson & Nunn, 2015).

Reviewing the phylogenetic analyses of the evolution of mammalian sleep, Nunn et al. (2016, p. 223) listed several important findings:

> First, predation risk appears to be a major predictor of sleep architecture in mammals, with safer options for sleep leading to more sleep. Similarly, animals at lower trophic levels (e.g., herbivores) sleep less than those at higher trophic levels (e.g., carnivores). Second, relative brain mass shows no association with sleep durations but does covary positively with the percentage of REM sleep across mammals; specific brain regions were also generally unrelated to sleep architecture, with the exception of the amygdala and NREM sleep (Capellini et al., 2009). Third, basal metabolic rate (with and without control for body mass) showed a negative association with sleep durations, suggesting that greater metabolic needs in a lineage favor less sleep. Fourth, animals with longer gestation lengths, sleep less, even after controlling for body mass. Fifth, species with more sleep have higher white blood cell counts and possibly fewer parasites (Preston et al., 2009). Finally, the durations of REM and NREM sleep covary positively (Capellini et al., 2010).

In summary, the total amount of time reserved for different sleep stages does not correspond with specific functional benefits related to these stages (Nunn et al., (2016). Furthermore, animals may have increased their REM and NREM sleep durations if ecological conditions allow them to sleep more, meaning that evolution regulates sleep duration depending on the costs and benefits of being awake, such as

predation, foraging, and social interactions. As Nunn et al. (2016, p. 233) stated: "…If an animal has something better to do than sleep (such as forage, court potential mates, or watch for predators), natural selection will favor shorter sleep durations.".

Today, post-industrial people are protected from most of the ancestral threats, but reduced sleep length and quality are still significant problems for many people. This suggests that new kinds of threats have become predominant in the present day, such as ostracism, loss of status, and low self-esteem (Perogamvros et al., 2020). It is certain that insomnia is a source of significant distress, causing impairments in daytime functioning and negative health outcomes in the long term. However, the evolutionary perspective raises the question of whether there could be some advantages of insomnia in terms of survival or reproduction.

In their evolutionary analysis of insomnia, McNamara and Auerbach (2001) drew attention to three important points. First of all, when we compare insomniacs to people with sleep deprivation, the latter are characterized by significant sleepiness on the following day. Although insomniacs subjectively report sleepiness the next day, the objective measures of daytime sleepiness do not indicate significant differences between the sleepiness of insomniacs and non-insomniacs (Chambers & Keller, 1993; Gumenyuk et al., 2015). It could be argued that insomniacs have a resistance to the homeostatic drive toward sleepiness, and they are similar to short sleepers (people requiring a small amount of sleep to be healthy and functioning) (McNamara & Auerbach, 2001).

Secondly, insomniacs in sleep medicine clinics generally regard the symptoms of stress as an outcome of insomnia rather than thinking that stress is the trigger of sleep disturbances (McNamara & Auerbach, 2001). It is obvious that insomnia further exacerbates the distress of the person, but in most cases, the precipitating factor of insomnia is a significant stressor (Perogamvros et al., 2020). The stressors arouse anxiety, which subsequently leads to hyperarousal. It is possible to explain the sleeplessness of insomniacs in terms of the hyperarousal of subcortical structures and

amygdalar circuits (Carr et al., 2003; Desseilles et al., 2008; McNamara & Auerbach, 2001). Thirdly, despite having daytime impairments of functioning in some areas, insomniacs actually preserve many essential cognitive functions, such as general cognitive functioning, perceptual processes, psychomotor functions, verbal functions, alertness, divided attention, sustained attention, vigilance, procedural memory, cognitive flexibility, and verbal fluency (Fortier-Brochu et al., 2012). To summarize, an insomniac can be defined as a relatively well-functioning person who develops resistance to the homeostatic drive toward sleepiness due to significant stressors related to survival or reproduction. In other words, an insomniac sacrifices the opportunity to sleep in order to deal with certain threats. As McNamara and Auerbach noted (2001, p. 114):

> The culprit in this whole story is, of course, anxiety. Anxiety leads to hyperarousal, which leads to insomnia. Why would it benefit an organism to restrict its time asleep and most especially restrict its time in slow-wave sleep when it is anxious? The simplest answer, of course, is that anxiety signals danger, and thus vigilance levels need to be maintained until the danger is past. After the danger is past, vigilance can be relaxed and lost sleep made up through the compensatory rebound process described previously.

In the case of insomnia, both REM and NREM sleep durations decrease, which may seem counterintuitive at first glance. During REM sleep, the prefrontal cortex is in a state of hypoarousal and cannot deal with environmental threats. Furthermore, the sleeper is paralyzed, cannot escape external threats, and therefore is more vulnerable to an external threat during REM sleep. According to McNamara and Auerbach (2001), if vigilance against a threat leads to insomnia, it is more advantageous to inhibit REM sleep instead of non-REM sleep. Yet, this is not the case. The answer to this dilemma could be found in other characteristics of REM sleep. Findings also indicated that aggression circuits of the brain show heightened activation, and there is a higher frequency of aggressive dream themes in REM sleep. Therefore, McNamara and Auerbach (2001) proposed that the anticipated response to a threat may be “fight” rather than “flight” during REM sleep. Thus, perhaps REM sleep is not inhibited due to its fight function.

From an evolutionary perspective, it can be proposed that acute insomnia that is characterized by hyperarousal and elevated vigilance, functions as an adaptive response to real or perceived stressors through the activation of the "fight or flight" system (McNamara & Auerbach, 2001; Perogamvros et al., 2020). Several lines of evidence supported the adaptive function of acute insomnia (Capellini et al., 2010; Haig, 2014; Hill et al., 2016; Lesku et al., 2008; Perlis et al., 2020; Vargas et al., 2020). Although acute insomnia seems to be adaptive against short-term ancestral threats, the adaptive functions of chronic insomnia are not as obvious. Perogamvros et al. (2020) argued that acute insomnia was adaptive in human history for fear eliciting ancestral threats, which were most of the time real and actual. But, modern-day threats are generally related to anticipated threats and are experienced as anxiety (Perogamvros et al., 2020). Considering evolutionary mismatch theory, which argues that previous adaptations dealing with past ancestral threats may be maladaptive in the modern context (Li et al., 2018), Perogamvros et al. (2020, pp. 4–5) stated:

> Chronic insomnia seems to reflect a pathological process where hyperarousal becomes self-perpetuating (via conditioning), even in the absence of the initial threat. In the framework of our evolutionary-emotional hypothesis, this translates into a failure of the fear extinction processes, with no decline in conditioned fear responses throughout time in certain vulnerable individuals.

Acknowledging the connections between environmental threats, fear responses, and acute insomnia, Perogamvros et al. (2020) asserted that hyperarousal and wakefulness in an acute insomnia period, which is perpetuated by fear conditioning, could turn into chronic insomnia due to failure of fear extinction even after threats disappear. In other words, while acute insomnia brings adaptive advantages in the case of real and actual threats, the same adaptive processes may become maladaptive in the future-oriented threats of modern times due to failure of fear extinction. Perogamvros et al. (2020, p. 5) stated:

> …when the initial stressor (US) that caused acute insomnia is absent, and if fear conditioning (CS/CR) has taken place during a 'sensitive' period…specific CS (e.g., bed, place, etc) can maintain CR (conditioned

wakefulness/arousal) for long periods and explain chronic insomnia. These stimuli should normally be compatible with sleep, but they become incompatible due to their association with the US.

Recent evidence indicated that some chronic insomniacs have heightened fear conditioning and delay or failure of fear extinction, supporting the proposition of Perogamvros et al. (2020) (Seo et al., 2018; Wassing et al., 2019). Thus, for insomniacs, conditioned stimuli, such as a bed, bedroom, or bedtime may evoke wakefulness due to fear conditioning, which associates these neutral stimuli with an unconditioned stimulus (stressor) and an unconditioned response (fear arousal). These associations are maintained in insomniacs because of delay or failure of fear extinction, finally causing chronic insomnia (Perogamvros et al., 2020). Based on the evolutionary insomnia model of Perogamvros et al. (2020), this thesis aimed to extend the model by asserting that IVI related to worry and ruminations during the pre-sleep period may produce fear conditioning, and this may be one of the reasons behind delayed or failed fear extinction in insomnia.

1.10 Fear Simulation Model of Insomnia (FSMI)

Given that threats related to survival and reproduction decrease evolutionary fitness (how well an organism survives and reproduces), we are the offspring of humans who developed certain responses to deal with ancestral threats. A threat in the evolutionary sense could be the loss of a relationship, property, social status or reputation, skill, abilities as well as one's life. (Marks & Nesse, 1994). The perception of these threats drives us to certain survival responses such as fight, flight, or freeze (Bracha, 2004). Mediating survival responses, the emotion of fear is triggered when a subject detects a threat, and it facilitates escaping from threats or preparing defensive responses (Maack et al., 2015). In this sense, fear could be regarded as an emotional response that occurs after encountering a negative stimulus (Öhman, 1993). In their factor analysis of 200 studies, Arrindel et al. (1991) found that there were four primary fear factors (1) fears regarding interpersonal events or situations (fear of criticism, rejection, and conflicts), (2) fears of death, injuries, illness, blood, and surgical

procedures; (3) animal phobia; (4) agoraphobia. All of these threats can produce fear responses in humans.

Overlapping with fear, anxiety is also a response given to threats by humans. However, in anxiety, the person anticipates threats long before meeting them, and the anxiety response is triggered before future threats occur (Epstein, 1972; Öhman, 1993). The anticipation of future threats and anxiety may lead to IVI of anticipated negative events (Hirsch & Holmes, 2007; Ji et al., 2016). I propose that IVI of past and future threats in the pre-sleep period simulate a fear response to these past or anticipated threats and that simulation process eventually leads to fear conditioning. For cases of acute insomnia, there may be two possibilities of threats that evoke arousal and therefore impair normal sleep. The first one is that there could be an actual and real threat, such as predators or social hostility (Perogamvros et al., 2020). The second possibility is that these threats can be anticipated or remembered in the form of IVI. It can be asserted that some individuals are able to simulate possible threats more realistically or vividly than others during bedtime as they worry or ruminate in the form of IVI, which can induce fear-arousal and conditioning (Dadds et al., 1997; Ji et al., 2016; Mertens et al., 2020).

It could be either that some people engage in IVI more frequently and/or have superior VIA, which makes their simulations more realistic and, as a result they experience stronger fear response. Thus, the actual threat or IVI of the potential threat evokes physiological and cognitive arousals, which lead to psychophysiological arousal impairing normal sleep. In cases of chronic insomnia, besides the fear conditioning formed during the acute insomnia phase due to real or perceived threats (Perogamvros et al., 2020), worry and rumination in the form of IVI (US) during and after an acute insomnia period constantly conditions sleep, bed, bedtime, bedroom (CS) with fear-arousal (UR). Consequently, IVI may prevent the possibility of fear extinction in the absence of real threats because fear extinction requires the formation of new CS-NoUS associations other than previous fear memory (Milad & Quirk, 2012; Myers & Davis, 2007). After the formation of fear learning, sleep-related

stimuli can readily evoke arousal in the absence of actual or simulated threats. Finally, the unresolved and prolonged fear responses activate a psychophysiological survival model characterized by hyperarousal and chronic insomnia symptoms.

FSMI may contribute to some of the six problematic points about insomnia models raised by Perlis et al. (2016). The first one is about the role of classical conditioning in insomnia disorder. FSMI directly emphasizes the importance of classical conditioning in the initiation and maintenance of insomnia as it argues that IVI may lead to the association of sleep-related stimuli with fear arousal. The second point refers to the effects of normal sleep-wake regulation on insomnia. FSMI does not include normal sleep-wake regulation by sleep homeostasis and circadian rhythmicity in its etiological explanations. These concepts can be integrated into the model. The third one is related to the differences between acute and chronic insomnia. Similar to the evolutionary-emotional hypothesis of insomnia (Perogamvros et al., 2020), FSMI regards acute insomnia as an adaptative response to real or perceived stressors and chronic insomnia as a negative byproduct of delayed or inhibited fear extinction.

The fourth point pertains to gender and age differences among insomnia patients. FSMI may provide an answer to the higher prevalence of chronic insomnia in women. Previous studies suggest that sex differences in brain systems, molecular pathways involved in fear extinction, gonadal hormones, and their interaction with the menstrual cycle may impair women's fear extinction (Velasco et al., 2019). Furthermore, having more vivid imagery (Campos & Pérez, 1988; Campos & Sueiro, 1993; Wassell et al., 2015; White et al., 1977), females may be more vulnerable to the effects of pre-sleep IVI on pre-sleep arousal (PSA). Finally, two previous studies found that females had higher intrusive thoughts scores than males (Ryckman & Lambert, 2015; Wegner & Zanakos, 1994). All these factors may lead to a higher prevalence of chronic insomnia in women. The fifth point is about the types and subtypes of insomnia. FSMI strives to explain primary insomnia but does not directly distinguish between sleep-onset, sleep-maintenance, and early morning awakening insomnias. Previous studies showed that PSA was significantly associated with sleep-

onset, sleep maintenance, and early morning awakening insomnia symptoms (Jansson-Fröjmark & Norell-Clarke, 2012; Ruivo Marques et al., 2018; Wuyts et al., 2012; Yeh et al., 2015). The sizes of correlations and expected R^2 values suggest that decreasing PSA evoked by IVI may help alleviate all three types of insomnia symptoms if the causality between IVI, PSA, and these insomnia symptoms are established by further studies. The final point is related to the role of elevated arousal or hyperarousal. FSMI assumes that hyperarousal in acute insomnia and chronic insomnia are physiologically or psychologically similar. However, further work is required to establish the validity of this assumption (Vargas et al., 2020).

FSMI is based on the evolutionary model of Perogamvros et al. (2020), but it expands the model of Perogamvros et al. in several ways. First of all, FSMI asserts that fear conditioning can occur via IVI both during and after the acute insomnia period. Second, as a result of the first proposition, constant conditioning of sleep-related stimuli with arousal (reconsolidation) may provide a reason for delay or failure of fear extinction. Third, VIA of the individual may be a precipitating factor for insomnia, meaning that the vividness of imagery may enhance the strength of the fear conditioning. The summary of this model can be seen in Figure 1. FSMI is based on three fundamental assumptions. First, IVI is able to produce a visual experience that is similar to its real-world equivalent, visual perception. Second, mental imagery can substitute real-life unconditioned stimulus (actual threats) in fear conditioning. Third, IVI must be present in insomnia. A variety of studies support these assumptions. Before presenting the evidence regarding the assumptions, the relationship between visual perception and imagery must be outlined.

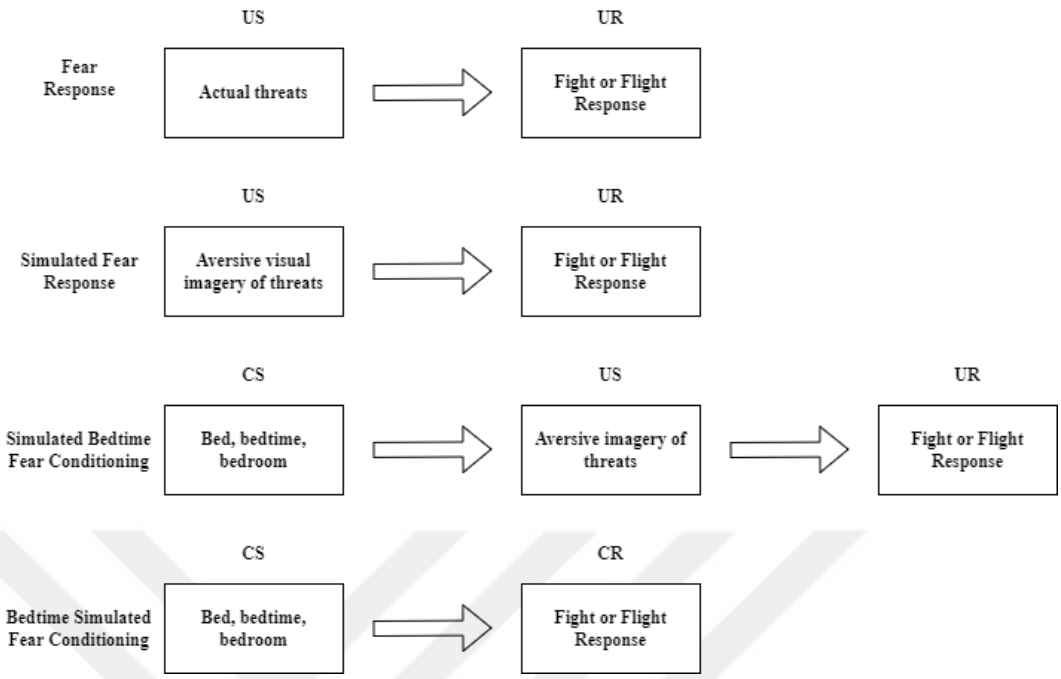


Figure 1 Fear Simulation Model of Insomnia

1.11 Visual Imagery and Visual Imagery Ability

Pearson et al. (2015, p. 591) described mental imagery as "representations and the accompanying experience of sensory information without a direct external stimulus." Visual mental imagery (VMI) refers to mental experiences that are defined as "seeing with the mind's eye" (Kosslyn et al., 2001, p. 635) that can emerge in the absence of an external stimulus (Ji et al., 2016). It is hypothesized that visual mental imagery is formed as the visual memories or representations stored in one's long-term memory are recalled and combined into novel inner visual stimuli (Ganis, 2013; Pearson et al., 2015; Pearson, 2019a). One's ability to form visual mental images is called visual imagery ability (Hall et al., 1985). According to Cumming and Eaves (2018), VIA consists of four main processes: image generation, image inspection, image transformation, and image maintenance. Image generation means creating images based on either retained sensory input or visual information stored in one's long-term memory. Image inspection refers to scanning, interpreting, and making sense of different parts of an image. Image transformation involves manipulating the content

or features of an image. Finally, image maintenance alludes to retaining a mental image during the processes of generation, inspection, and transformation.

VIA has three different qualities: vividness, controllability, and preference (Richardson, 1994). Vividness can be defined as "the clarity, sharpness, and sensory richness of the image" (Cumming & Eaves, 2018, p. 6). Controllability refers to the ability to manipulate images. Preference implies a person's preferred or dominant sensory modality, which could be either visile, audile, or motile (Richardson, 1994). Previous research conducted with self-reports or objective measures of visual imagery has indicated that there are individual differences in terms of vividness, controllability, and preference (Cui et al., 2007; Galton, 1880; Reeder, 2017; Richardson, 1994). For example, it was found that females had more vivid visual imagery than males (Campos & Pérez, 1988; Campos & Sueiro, 1993; Wassell et al., 2015; White et al., 1977), while males obtained higher scores on imagery manipulation performance test than females (Campos, 2014; Campos et al., 2004). In terms of preference, recent research yielded that individuals can be categorized into three groups: object-visualizers, spatial-visualizers, and verbalizers (Blazhenkova & Kozhevnikov, 2009; Shin & Kim, 2015). Perez-Fabello et al. (2018) compared 99 fine arts, 92 psychology, and 90 engineering students in terms of object, spatial, and verbal cognitive styles. It was found that the fine arts and psychology students preferred object imagery, while the engineering students were more likely to use spatial imagery. In terms of verbal cognitive style, the psychology students had higher scores than the fine arts and engineering students.

In the present study, it was argued that the level of VIA might be an important factor contributing to fear conditioning underlying insomnia. A person with higher VIA may simulate past and future threats more realistically than a person with lower VIA, and therefore may have stronger physiological responses to IVI and experience heightened fear conditioning. Thus, the moderator role of VIA on the relationship between IVI and PSA was investigated in Study 2. Although the model that was investigated in the present study is a model based on classical conditioning, it is more

detailed than a typical classical conditioning model with the addition of variables such as IVI and VIA. Up to now, no previous studies have investigated the role of VIA in insomnia.

1.12 Visual Imagery and Its Relationship with Visual Perception

It has been proposed that visual imagery imitates visual perception (VP) in many aspects or can be regarded as a weaker form of it (Pearson et al., 2015). There are several intriguing studies supporting this hypothesis. For instance, Laeng and Sulutvedt (2014) demonstrated that eye pupils respond to even the changes in brightness of imagery light. The study of Tartaglia et al. (2009) indicated that imagining a vertical line between two actual lines enhanced the performance of assessing distances between three perceptual lines, meaning that visual mental imagery facilitates perceptual learning. In their study with two unilateral neglect patients who cannot detect or respond to stimulus on one side of the visual area due to damage in one hemisphere of the brain (Bisiach et al., 1996), Bissiac and Luzzatti (1978) reported that the patients neglect one side of space when they are asked to imagine being in front of a famous landmark and describe the perceptual scene around it. This finding suggests that a neuropsychological condition affecting visual perception also affects visual imagery, meaning that the same brain areas may process both VP and VMI.

The recent developments in neuroscience have enabled researchers to distinguish which brain areas are activated in particular cognitive processes and test whether the processes regarding VP and VMI utilize the same brain areas. In a preliminary positron emission tomography study with six participants, Kosslyn et al. (1997) found that the same 14 out of 21 brain areas were activated when the participants engaged in VP and VMI. Testing the hypothesis that imagery and perception share common processes and representations, Ganis et al. (2004) conducted an fMRI study with 15 participants. In the imagery condition, the participants were asked to visualize objects via headphones, while in the visual perception condition, they were presented with pictures of the objects. It was found that there were extensive overlaps between the

brain areas processing VP and VMI. The results also indicated that brain areas processing VMI were a subset of those which were processing VP.

Laeng and Teodorescu (2002) investigated the eye movements of participants in visual perception and imagery conditions. The results revealed that there were positive linear relationships between the eye movements of the participants in both conditions. In their fMRI study with 8 participants, Cui et al. (2007) showed that there was a strong correlation between visual cortex activity during imagery and the scores from the Vividness of Visual Imagery Questionnaire (VVIQ) ($r = -.73$). The relationship between visual cortex activity and the self-reported vividness of imagery was also supported by Dijkstra et al. (2017) in terms of the fluctuation of vividness between trials. In their fMRI study with 11 participants, Lee et al. (2012) found that the identity of seen and imagined objects could be decoded from the pattern of activity in the ventral visual processing stream, meaning that perception and imagery share similar neural substrates. Similarly, Albers et al. (2013) were able to distinguish the kind of stimulus from neural activity patterns of the visual cortex during VMI. They found that these activity patterns were similar to those evoked by visual perception (Albers et al., 2013). To conclude, VMI can activate the majority of the brain regions that are activated during visual perception, and the neural activities of VMI mostly mimic the neural activities of VP. These findings indicate that VMI may evoke emotional and physiological responses similar to VP.

1.13 The Effects of Visual Imagery and Visual Imagery Induced Fear Conditioning

Another critical issue is that despite similarities between the brain activities related to VP and VMI, whether VMI can produce cognitive and physiological arousal and substitute the real US in fear conditioning. Studies conducted over the past forty years yielded three important findings. First, the VMI of stimulus can produce psychophysiological arousal, such as heart rate acceleration, elevated skin conductance, elevated startle blink reflex, and an increase in respiratory rate (Ji et al., 2016; Lang, 1979). Second, the physiological effects induced by the VMI of stimulus

were higher than the effects of verbal processing of stimulus (Holmes et al., 2008; Holmes & Mathews, 2005; Vrana et al., 1986). Third, the higher vividness of imagery was associated with higher arousal (Cui et al., 2007; Ji et al., 2016; McNeil et al., 1993; Rauch et al., 2004).

Moreover, a great deal of previous research has established that the VMI of CS, US, or CS-US contingency can function similar to the actual administration of CS, US or CS-US contingency in fear conditioning (Dadds et al., 1997; Mertens et al., 2020). For example, imagined extinction can reduce physiological responses to threatening cues and activate the brain areas that were found to be active in actual extinction (Agren et al., 2017; Grégoire & Greening, 2019; Reddan et al., 2018). Similarly, the imagination of US or CS-US can substitute the actual administrations in fear conditioning (Joos et al., 2012; Krypotos et al., 2019; Mueller et al., 2019). Finally, the imagination of fear associations may exacerbate learned fear (Arntz et al., 1997; Davey & Matchett, 1994). In conclusion, the physiological effects of the VMI of stimulus and the way it can lead to fear conditioning suggest that intrusive thoughts comprising visual imagery can have similar effects during the sleep-onset period. The evidence also indicates that more powerful VIA leads to a stronger effect of VMI. Therefore, in Study 2, the indirect effects of IVI and intrusive verbal thoughts (IVT) on insomnia severity through PSA were compared. Moreover, the moderator role of VIA in the relationship between IVI and PSA was investigated.

1.14 Intrusive Thoughts

Although VMI can be intentional when people exercise their VIA, it can also be intrusive and uncontrollable. The concept of intrusive thoughts was introduced by Rachman and Silva (1978) as an etiological factor contributing to obsessive-compulsive disorder. Intrusive thoughts refer to recurring thoughts, images, or impulses that are experienced as unacceptable and unwanted by the person (Clark & Purdon, 1995; Rachman, 1978). Rachman (1981) listed the three important features of an intrusive thought: its disruption of an ongoing activity, its internal origin, and difficulty in controlling it. Similarly, Julien et al. (2007) regarded intrusive thoughts,

images, and impulses as unwanted, repetitive, and intrusive. The content of intrusive thoughts can be disgusting, blasphemous, immoral, or worrisome (Parkinson & Rachman, 1981).

In their pioneer study, Rachman and Silva (1978) investigated whether people who do not have a psychiatric disorder experience intrusive thoughts and the differences between intrusive thoughts and obsessions. The results showed that 79.84% of the non-clinical participants reported having intrusive thoughts. Further comparison between obsessional patients and non-clinical participants revealed that intrusive thoughts and obsessions were similar in terms of content (harm to self and others, socially unacceptable behaviors, sexual or aggressive acts, health concerns), their effect on the person's mood (discomfort), and meaningfulness (Rachman & de Silva, 1978). On the other hand, obsessions were characterized by being more acceptable, being harder to dismiss, having lower controllability, longer duration, higher subjective discomfort and intensity, higher frequency, and provoking more urge to neutralize compared to intrusive thoughts (Rachman & de Silva, 1978).

In a following study with 60 participants, Parkinson and Rachman (1981) reported that the majority of the participants experienced intrusive thoughts. While 86% of participants stated having intrusive thoughts, 66.6% reported that they have intrusive images. The comparison of intrusive thoughts, images, and impulses yielded that intrusive impulses were more intense, stressful, and frequent than intrusive thoughts and images. While intrusive images were less acceptable and shorter in duration, intrusive thoughts were harder to control in comparison to intrusive images (Parkinson & Rachman, 1981). These findings indicated that different types of intrusive thoughts may have different effects on individuals' experiences. Although intrusive thoughts in non-clinical populations are experienced less frequently compared to clinical obsessions (Purdon & Clark, 1994), intrusive thoughts are also experienced by non-clinical subjects (Freeston et al., 1991; Julien et al., 2007; Purdon & Clark, 1993; E. Watkins et al., 2005; Wells & Morrison, 1994). Therefore, researchers argued that what transforms normal intrusive thoughts into obsessional

cognitions may be obsessional patients' interpretations and appraisals of these thoughts (Clark & Purdon, 1995; Rachman, 1993; Salkovskis et al., 1995).

Intrusive thoughts can also elicit physiological responses that may bring adverse health consequences, especially in the long term. It has previously been observed that intrusive thoughts are positively associated with skin conductance, heart rate, heart rate variability, cortisol levels, and central nervous system arousal, meaning that intrusive thought may alter cardiovascular, autonomic, and endocrine system activities (Aldao et al., 2013; Hofmann et al., 2005; Linz et al., 2018; MacNeil et al., 2017; Ottaviani et al., 2016; Pillai & Drake, 2015; Rydstedt et al., 2011; Weise et al., 2013). In their meta-analysis of experimental studies examining the relationship between perseverative cognitions (rumination and worry) and physiological variables, Ottaviani et al. (2016) found the effect sizes of the effects of perseverative cognitions on physiological variables as g = .45 for systolic blood pressure, g = .51 for diastolic blood pressure, g = . 28 for heart rate, g = .15 for heart rate variability, and g = .36 for cortisol levels.

Despite the fact that most of the previous studies examined the relationship between intrusive thoughts and obsessive-compulsive disorder, the evidence yielded that intrusive thoughts are also present in depression (Moritz et al., 2019; Wenzlaff, 2002), generalized anxiety disorder (Gross & Eifert, 1990), hypochondria (Arnáez et al., 2021; Romero-Sanchiz et al., 2017), and post-traumatic stress disorder (Falsetti et al., 2002; Hagenaars et al., 2010; Nixon et al., 2021). Another disorder that intrusive thoughts seem to have an important role in is insomnia (Clark, 2004). Intrusive thoughts in the sleep-onset period impede insomniacs' ability to fall asleep by preventing automatic inhibition of wakefulness (Baglioni et al., 2010; Pillai & Drake, 2015). As Borkovec (1982, p. 892) stated: "A large number of insomnias are due to intrusive, relatively uncontrollable, cognitive activity, especially what might be best labeled as worry.". Three studies by Harvey (2002), Watts et al. (1994), and Wicklow and Espie (2000) indicated that the contents of intrusive thoughts before falling asleep consist of rehearsals, problem-solving, long-term concerns, worries

about sleep and its consequences, somatic preoccupations, arousal status, focusing on external noises, and focusing on time. Previous research has established that intrusive thoughts and pre-sleep cognitive activity were associated with shorter sleep length (Kelly, 2002; Nota & Coles, 2015), increased sleep-onset latency (Nicassio et al., 1985; Nota & Coles, 2015; Wicklow & Espie, 2000), and decreased sleep quality (Baker et al., 2015; M. Hall et al., 2000).

There are also several experimental studies that investigated the effects of anxiety-inducing manipulation, which is believed to increase intrusive thoughts, on sleep parameters. In their study with 38 female good sleepers, Gross and Borkovec (1982) instructed the manipulation group that they would give a speech about how to reduce inflation without causing an economic depression just before they take their afternoon naps. Compared to two control groups, one of which was also told that they would give a speech, but without a predetermined topic, it took longer for the manipulation group to fall asleep. Lichstein and Fanning (1990) examined the sleep-onset period skin conductance of 15 insomniacs and 15 good sleepers who were told that a polygraph malfunction might accidentally shock them. The results showed that insomniacs had significantly higher skin conductance responses, which is associated with cognitive arousal, as they strive to sleep compared to good sleepers. In their study, Hall et al. (1996) compared two groups of students who were told that they would either give a speech or read magazines the next morning. It was found that the speech group experienced more intrusive thoughts pertaining to the task, and it took longer for them to fall asleep compared to the magazine group. Finally, Tang and Harvey (2004) examined the effects of anxious and neutral cognitive arousal on sleep perceptions in a sample of 54 good sleepers. The first group was instructed to give a speech the next day, the second group was asked to write an essay on waking, and the third group did not receive any manipulation. It was found that only the first and second groups experienced increased cognitive activity and had longer sleep-onset latency.

Although the term intrusive thoughts seem to refer to a single construct, an intrusive thought could be a verbal thought, image, or impulse, all of which may have different effects on one's emotional experience. The Bio-informational theory developed by Lang (1979) asserts that "…a mental imagery representation of an emotionally charged stimulus (e.g., a spider) activates an associative network of stored information that overlaps with that activated during actual experience of the stimulus in reality (e.g., encountering a live spider)…" (Ji et al., 2016, p. 703). Building on Lang's bio-informational theory, Hagenaars et al. (2010) emphasized the importance of distinguishing the effects of IVI and IVT on emotions and physiological arousal. As suggested by previous studies, IVI may have a more profound effect on emotions and physiological arousal due to an activation of the visual cortex and an induction of fear conditioning as compared to IVT (Holmes et al., 2008; Holmes & Mathews, 2005; Ji et al., 2016; Lang, 1979; Vrana et al., 1986), meaning that visual imagery may play a significant role in the etiology of psychopathologies rather than only being an autobiographical memory function (Brewin et al., 2010; Hagenaars & Holmes, 2012).

Several studies have shown that IVI is present in insomnia. To test the hypothesis that thinking in images facilitates emotional processing of worry better than thinking in VTs (Borkovec et al., 1998), Nelson and Harvey (2002) compared two groups of insomnia patients who were asked to think about a five-minute speech that they would give the following day either in images (n = 14) or in verbal thoughts (n = 17) before sleep. It was revealed that the imagery group reported higher distress and arousal and had lower subjective sleep-onset latency following the experimental manipulation than the verbal group. There were no differences between the groups in terms of objective sleep-onset latency durations. In addition, the imagery group was less anxious and regarded their thoughts about the speech as being less uncomfortable the next morning compared to the verbal thoughts group, suggesting more successful emotional processing for imagery groups in the long term. In terms of the vividness of visual imagery, there was not a significant difference between the VVIQ scores of the two groups. Interpreting the results, the authors commented that imagery

techniques facilitating emotional processing by increasing thinking in images and reducing thinking in VTs in the pre-sleep period might be an intervention for insomnia (Nelson & Harvey, 2002). Compared to these findings, the present study argues that involuntary and uncontrollable visual imagery may have detrimental effects on sleep.

In their follow-up study, Nelson and Harvey (2003a) assessed the relationship between unpleasant images and sleep-onset latency in insomniacs (n = 20) and good sleepers (n = 20). The results showed that insomniacs had more unpleasant images and less pleasant images than good sleepers in the pre-sleep period, even after controlling for sleep-onset latency. Again, there was no significant difference between the two groups in regard to VVIQ scores (Nelson & Harvey, 2003a). Finally, Nelson and Harvey (2003b) investigated the pre-sleep images and VTs before an afternoon nap in sleep-onset insomnia patients (n = 34) and good sleepers (n = 38). The participants in the insomnia group reported significantly more negative images than positive images compared to good sleepers, and all participants rated negative images as being less controllable than negative verbal thoughts. In terms of the vividness of the visual imagery, there was not a significant difference between the VVIQ scores of the two groups (Nelson & Harvey, 2003b). Taken together, these studies supported the notion that VMI may be a factor aggravating insomnia symptoms. In the case of insomnia, it could be argued that IVI during the sleep-onset period leads to higher PSA, which is a precursor for insomnia, compared to IVT. In Study 2, IVI and VTs were included in the path model as predictors of PSA.

1.15 Pre-sleep Arousal

Previous research and proposed insomnia models have suggested that physiological and cognitive hyperarousal contributes to the predisposition to and perpetuation of insomnia disorder (Marques et al., 2015; Nicassio et al., 1985; Perlis et al., 2016; Riemann et al., 2010). It was hypothesized that the hyper-aroused state of insomniacs in the pre-sleep period interferes with the initiation and maintenance of sleep (Bastien, 2011) as though the sleep system is suspended or disrupted by the

inappropriate activation of the central nervous system (Bonnet & Arand, 2010). In the context of insomnia research, PSA generally refers to somatic and cognitive arousals (Broman & Hetta, 1994b; Nicassio et al., 1985; Riemann et al., 2010). Somatic arousal refers to the psychosomatic responses given to a threat-related stimuli and is characterized by increased heart rate, respiration rate, muscle tension, glucose release, perspiration, and dilation of the pupils (Nicassio et al., 1985; Vargas et al., 2020). Cognitive arousal pertains to pre-sleep experiences of excessive negatively-toned mental activities related to worrying and rumination, having a racing mind, and not being able to shut off one's thoughts (Broman & Hetta, 1994b; Lemyre et al., 2020; Nicassio et al., 1985; Puzino, Amatrudo, et al., 2019).

Up until to now, the evidence has indicated that insomnia patients have higher pre-sleep cognitive and somatic arousal scores compared to good sleepers, and higher PSA is associated with longer sleep-onset latency, decreased total sleep time, more frequent awakenings from sleep, more frequent early morning awaking, decreased sleep quality, higher daytime impairment, and higher insomnia severity (Broman & Hetta, 1994b; Jansson-Fröjmark & Norell-Clarke, 2012; Marques et al., 2015; Nicassio et al., 1985; Palagini et al., 2016, 2017; Puzino, Amatrudo, et al., 2019; Ruivo Marques et al., 2018; Yeh et al., 2015).

Several studies have examined the relationship between intrusive thoughts, PSA, and sleep quality. Yeh et al. (2015) conducted a study with 202 undergraduate students. The results of the structural equation modeling showed that higher repetitive thoughts (dwelling on the negative and worry engagement) predicted higher PSA, and higher PSA predicted lower sleep quality, yielding a mediator role for PSA in the relationship between repetitive thoughts and sleep quality. Two more recent studies investigated the moderator role of intrusive thoughts on the relationship between psychological stress and sleep problems (Benham, 2021; Tousignant et al., 2019). In their study with 178 participants, Tousignant et al., (2019) showed that rumination and worry (two common forms of intrusive thoughts) were moderators of the relationship between stress and PSA, meaning that a higher level of intrusive thoughts

predicted a stronger relationship between stress and PSA. Similarly, Benham (2021) found that bedtime repetitive negative thinking was a significant moderator of the relationship between stress and insomnia, with higher repetitive negative thinking predicting a stronger relationship between stress and insomnia. Overall, the evidence suggests that PSA evoked by intrusive thoughts may deteriorate sleep and lead to insomnia. In the current study, PSA was used as a mediator variable through which IVI and IVT affected insomnia severity.

1.16 Present Study

The aim of the present study was to investigate whether IVI and IVT significantly predict PSA and insomnia severity and whether there is a moderator role of VIA on the relationship between IVI and PSA in a sample of people who meet the DSM-V criteria for insomnia disorder. The current study design could not directly address the fear conditioning hypothesis of chronic insomnia but aimed to provide preliminary evidence for the hypothesis. The model of the present study can be seen in Figure 2. In Study 1, Pre-sleep Arousal Scale and the Intrusive Visual Imagery and Verbal Thought Questionnaires were adapted to Turkish. In Study 2, the model comprising IVI, IVT, VIA, PSA, and insomnia severity was tested with path analysis.

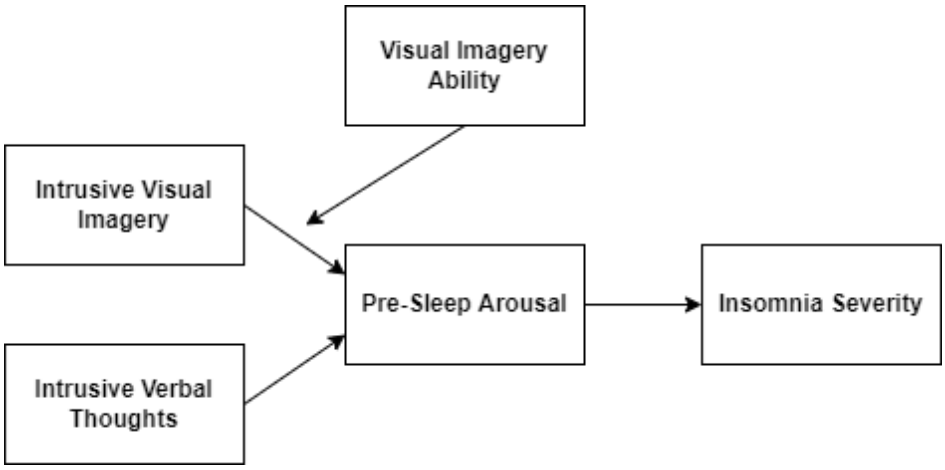


Figure 2 The Model of The Present Study

The specific hypotheses of the present study are:

1) IVI would significantly predict PSA.
2) IVT would significantly predict PSA.
3) The effect size of the path coefficient from IVI to PSA would be higher than the effect size of the path coefficient from IVT to PSA.
4) PSA would significantly predict insomnia severity.
5) VIA would moderate the relationship between IVI and PSA.

CHAPTER 2

STUDY 1: PSYCHOMETRIC PROPERTIES OF PRE-SLEEP AROUSAL SCALE AND INTRUSIVE VISUAL IMAGERY AND VERBAL THOUGHT QUESTIONNAIRES IN TURKISH SAMPLES

METHOD

2.1.1 Participants

Six hundred fifty-one participants participated in this study. The participants were recruited via social media and the METU Psychology department's online data collection system, SONA. To prevent unreliable results from overfitting (Fokkema & Greiff, 2017), the sample was split half in SPSS 25, and exploratory factor analyses and confirmatory factor analyses were conducted on separate samples. Convergent, divergent, incremental, and discriminant validities were tested on a subsample of 556 participants who completed all questionnaires in the study. Moreover, a third sample having 88 participants was used to conduct 3 weeks period test-retest analysis.

The mean ages of the participants for the four samples were 28.56 (*SD* = 14.11), 27.63 (*SD* = 13.89), 29.25 (*SD* = 14.81), and 22.19 (*SD* = 4.98), respectively. There were 207 females (63.50%) and 119 (35.89%) males in the first sample, 218 females (67.08%) and 107 males (32.92) in the second sample, 349 females (62.77%) and 207 males (37.23) in the subsample, and 71 females (80.68%) and 17 males (19.32%) in the third sample. The majority of the participants in the four samples had university-level education (88.96%, 88.31%, 86.69%, and 100%, respectively). 18.71% of the first sample, 18.77% of the second sample, 21.94% of the subsample, and no one in the third sample were employed. The majority of the participants in the first (77.61%), second (80.92%), sub (75.72), and third samples (94.31%) were single. 67.18% of the first sample, 66.46% of the second sample, 66.55% of the subsample,

and 64.77% of the third sample were from middle SES. The demographic characteristics of the participants can be seen in Table 3.

Table 3 Demographics for The Samples of Study 1

	First Sample		Second Sample		Subsample		Third Sample	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Age	28.56	14.11	27.63	13.89	29.25	14.81	22.19	4.98
	N	%	*N*	%	*N*	%	*N*	%
Gender								
Female	207	63.50	218	67.08	349	62.77	71	80.68
Male	119	35.89	107	32.92	207	37.23	17	19.32
Education								
High School	9	2.76	6	1.85	15	2.70	0	0
University	290	88.96	287	88.31	482	86.69	100	100
Graduate	27	8.28	32	9.85	59	10.61	0	0
Employment								
No	265	81.28	264	81.23	434	78.06	100	100
Yes	61	18.71	61	18.77	122	21.94	0	0
Marital Status								
Single	253	77.61	263	80.92	421	75.72	83	94.31
Married	69	21.17	57	17.54	126	22.66	5	5.68
Divorced	4	1.23	5	1.54	9	1.62	0	0
Socioeconomic Status								
Very Low	20	6.14	15	4.62	31	5.58	1	1.14
Low	47	14.42	47	14.46	80	14.39	10	11.36
Middle	219	67.18	216	66.46	370	66.55	57	64.77
High	39	11.96	46	14.15	73	13.13	9	21.59
Very High	1	.31	1	.31	2	.36	1	1.14

2.1.2 Instruments

2.1.2.1 Demographic Information Form

A demographic information form was used to assess the characteristic of participants. Questions asking about participants' age, gender, level of education, employment status, marital status, and socioeconomic status were included. The form can be seen in Appendix E.

2.1.2.2 Pre-sleep Arousal Scale

Pre-sleep Arousal Scale (PSAS-16) was developed by Nicassio et al. (1985) to measure the state of arousal before sleep. The scale consists of 16 items, which are rated on 5-point Likert scale, ranging from 1 (not at all) to 5 (extremely). PSAS-16 comprises two factors, each having eight items measuring cognitive (PSAS-C) and somatic (PSAS-S) arousal in the pre-sleep period. Sample items include "Review or ponder events of the day." and "Cold feeling in your hands, feet or your body." The scale development sample included 147 undergraduate students, 30 insomniacs, and 30 normal sleepers. The internal consistency coefficients for cognitive and somatic subscales were .88 and .79 for undergraduates, .81 and .76 for insomniacs, and .67 and .84 for normal sleepers. The test-retest analysis showed that the correlation between baseline and three weeks follow-up scores were .72 and .76 for cognitive and somatic subscales in a subgroup of 30 undergraduate students.

PSAS-C and PSAS-S had significant positive correlations with Taylor Manifest Anxiety Scale (r = .50 and r = 58, respectively) and the Center for Epidemiological Studies Depression Scale (r = .40 and r = .41, respectively) (Nicassio et al., 1985). While PSAS-C was only significantly correlated with the cognitive subscale of the Cognitive-Somatic Anxiety Questionnaire (r = .36), PSAS-S was significantly correlated with the cognitive and somatic subscales of the Cognitive-Somatic Anxiety Questionnaire (r = .49 and r = .52, respectively). Moreover, the somatic and cognitive subscales of PSAS-16 had small to high correlations with self-report sleep-related indices such as considering oneself as insomniac (r = .21 and r = .45, respectively), sleep-onset latency (r = .29 and r = .59, respectively), total sleep duration (r = -.19 and r = -.34, respectively), awakenings from sleep (r = .29 and r = .35, respectively), and listlessness during the day (r = .23 and r = .45). In the undergraduate sample, three groups who had varying sleep-onset latency (those less than 20 minutes, between 20 and 40 minutes, and more than 40 minutes) had significantly different scores from cognitive and somatic subscales of PSAS-16, showing that arousal levels elevate as sleep latency increases. A comparison of insomniacs and normal sleepers in terms of individual items of PSAS-16 yielded that insomniacs had significantly

higher scores from all items. In the second study of Nicassio et al. (1985), 31 undergraduate students completed PSAS-16 and sleep indices (sleep-onset latency, awakenings from sleep, total sleep time) every morning by rating the previous night over ten days. PSAS-C and PSAS-S had significant correlations only with sleep-onset latency (r_{Mean} = .42 and r_{Mean} = .32, respectively).

Jansson-Fröjmark & Norell-Clark (2012) examined the psychometric properties of PSAS-16 in a sample of 1890 participants from Sweden. In exploratory factor analysis, the two-factor structure was replicated, but three items of the cognitive subscale had low loadings, and they were excluded from further analysis. The Cronbach alpha coefficients of the total scale, cognitive and somatic factors were .85, .88, and .72, respectively. There were strong significant correlations between three items removed form of PSAS (PSAS-13) and PSAS-16 (ρ = .99) and three items removed form of PSAS-C (PSAS-C-5) and PSAS-C (ρ = .97). Regarding discriminant validity, both PSAS-13 and PSAS-16 significantly distinguished the insomnia group from the poor and normal sleepers and the poor sleepers from the normal sleepers. PSAS-13 and its subscales had significant correlations with Anxiety and Preoccupation about Sleep Questionnaire (ρ = .43-.52), Dysfunctional Beliefs and Attitudes about Sleep Scale (ρ = .38-.45), Hospital Anxiety and Depression Scale - Anxiety subscale (ρ = .49–.57), Hospital Anxiety and Depression Scale - Depression subscale (ρ = .38-.44). Finally, PSAS-13 and its subscales had significant correlations with sleep-onset latency (η = .26-.33), wake after sleep onset (η = .26-.31), early morning awakening (η = .21-.23), total sleep time (η = .20-.24), and sleep quality at a fair level (ρ = .35-.43). Previously, PSAS was adapted to German (Gieselmann et al., 2012), Pakistani (Namra & Tazvin, 2014), Portuguese (Ruivo Marques et al., 2018), and Japanese. In Study 1, PSAS-16 was adapted to Turkish.

2.1.2.3 Intrusive Visual Imagery and Verbal Thought Questionnaires

Intrusive Visual Imagery and Verbal Thought Questionnaires (IVIVTQ) were developed by McCarthy-Jones et al. (2012) to assess intrusive mental contents in the forms of visual imagery and verbal thoughts. Each questionnaire consists of 10 items

selected and adapted from the White Bear Suppression Inventory (Wegner & Zanakos, 1994) and the Thought Control Ability Questionnaire (Luciano et al., 2005). The items are rated on 5-point Likert scales ranging from 1 (Strongly disagree) to 5 (Strongly agree). Higher scores indicate higher levels of intrusive thoughts. Sample items are ‘‘My thoughts frequently return to one image.” and ‘‘There are verbal thoughts that keep jumping into my head.”.

Principal components factor analysis yielded a two-factor structure distinguishing imagery and verbal thoughts (McCarthy-Jones et al., 2012). The Cronbach’s alpha coefficients were .89 for intrusive visual imagery (IVIVTQ-Visual) and .90 for intrusive verbal thought (IVIVTQ-Verbal). The test-retest reliability analysis showed correlations between baseline and one-month follow-up scores to be .70 and .73 for IVIVTQ-Visual and IVIVTQ-Verbal, respectively. In terms of convergent validity, both IVIVTQ-Visual and IVIVTQ-Verbal had significant moderate correlations with Hospital Anxiety and Depression Scale - Anxiety subscale (r = .45 and .43, respectively), Hospital Anxiety and Depression Scale - Depression subscale (r = .30 and .31, respectively). While there was a significant medium correlation between IVIVTQ-Visual and Spontaneous Use of Imagery Scale (r = .43), IVIVTQ-Verbal was not significantly associated with the Spontaneous Use of Imagery Scale (r = .17). Moreover, IVIVTQ-Visual and IVIVTQ-Verbal were significantly correlated with PANAS - Negative affect (r = .36 and .45, respectively), but not with PANAS - Positive affect (r = -.04 and -.10, respectively). In terms of incremental validity, IVIVTQ-Visual and IVIVTQ-Verbal predicted hypomania scores after controlling for anxiety, depression, positive affect, and negative affect. In Study 1, IVIVTQ was also adapted to Turkish.

2.1.2.4 Beck Anxiety Inventory

Beck Anxiety Inventory (BAI) was developed by Beck et al. (1988) to assess the severity of anxiety symptoms. The scale has 21 items that are rated on 4-point Likert scale ranging from 0 (not at all) to 3 (seriously). Higher scores indicate higher levels of anxiety symptoms. Sample items include ‘‘Feeling hot’’ and ‘‘Unable to relax”.

BAI has two factors consisting of somatic (8 items) and subjective anxiety and panic (13 items) symptoms. Turkish adaptation of BAI was conducted by Ulusoy et al. (1998). The Cronbach's alpha of the Turkish version was found to be .93. Similar to the original scale; the Turkish form also had two factors. The scale was found to be correlated with the State-Trait Anxiety Inventory-Trait subscale (r = .45), State-Trait Anxiety Inventory-State subscale (r = .53), and Hopelessness Scale (r = .34), and Automatic Thoughts Questionnaire (r = .41), indicating its concurrent and divergent validities. In terms of discriminant validity, The Turkish form was able to distinguish anxious patients from depressed patients, other disorder patients, and control participants (Ulusoy et al., 1998). In Study 1, the somatic subscale of BAI was used to examine the convergent validity of PSAS-S. The internal consistency coefficient for the somatic subscale of BAI was .81 in the current study.

2.1.2.5 Ruminative Thought Style Questionnaire

Ruminative Thought Style Questionnaire (RTSQ) was developed by Brinker & Dozois (2009) to assess the ruminative thought style that is independent of depressive symptomology. The scale consists of 20 items measuring positive, negative, and neutral thoughts as well as past and future-oriented ruminative thoughts. RTSQ is rated on a 7-point Likert scale ranging from 1 (not at all) to 7 (very well). Higher scores on the scale indicate a higher ruminative thought style. Sample items read as ''I tend to replay past events as I would have liked them to happen'' and ''When I am looking forward to an exciting event, thoughts of it interfere with what I am working on". RTSQ was adapted to Turkish by Karatepe (2007). The Cronbach's alpha coefficient of the Turkish form was .91. The test-retest analysis showed that the correlation between baseline and one-month follow-up scores was .84. In terms of validity, the correlations between the Turkish version of RTSQ and Rumination, Comprehensibility, and Acceptance of Feelings subscales of the Leahy Emotional Schema Scale were .47, -.36, and -.14, respectively. Moreover, RTSQ had significant correlations with Beck Depression Inventory (r = .39) and Beck Anxiety Inventory (r = .44). In Study 1, the scale was used to assess the convergent validity of PSAS-C. The internal consistency coefficient for RTSQ was .96 in the current study.

2.1.2.6 Insomnia Severity Index

Insomnia Severity Index (ISI) was developed by Bastien et al. (2001) to provide a brief screening measure of insomnia severity. ISI has seven items covering the severity of sleep-initiating, sleep maintenance, early morning awakening problems, satisfaction with current sleep pattern, impairment in daily functioning, noticeability of impairment attributed to the sleep problem by other people, and general level of distress caused by the sleep problem. The subjects rate ISI on a 5-point scale. Higher scores indicate higher insomnia severity. ISI has a three-factor structure comprising impact, severity, and satisfaction subscales. The Turkish version of ISI was developed by Boysan and colleagues (2010). The internal consistency coefficient of the Turkish form was .79. Both exploratory and confirmatory factor analyses yielded that the Turkish form of ISI has two factors, namely daily functionality and sleep quality. In terms of validity, the Turkish version of ISI had moderate to high correlations with Beck Depression Inventory (r = .52), Hamilton Depression Scale (r = .48), and Pittsburg Sleep Quality Index (r = .64). In Study 1, the Turkish form of ISI was used to assess the incremental validity of PSAS-C and PSAS-S beyond depression, anxiety, and stress. The internal consistency coefficient for ISI was .80 in the current study.

2.1.2.7 The Object - Spatial Imagery and Verbal Questionnaire

The Object-Spatial Imagery and Verbal Questionnaire (OSIVQ) was developed by Blazhenkova and Kozhevnikov (2008) to assess object imagery, spatial imagery, and verbal cognitive styles. OSIVQ has a three-factor structure (object imagery, spatial imagery, and verbal) and 45 items. There are 15 items for each subscale. OSIVQ is rated on a 5-point Likert scale ranging from 1 (disagree) to 5 (agree). Sample items include “When I imagine the face of a friend, I have a perfectly clear and bright image.”, “My verbal abilities would make a career in language arts relatively easy for me.” and “I have excellent abilities in technical graphics.”. The Turkish version of OSIVQ was developed by Nuhoğlu and Akkoyunlu (2012). Confirmatory factor analysis revealed that the three-factor Turkish form of OSIVQ fit the data well, similar to the original scale. However, two items from the verbal subscale were

excluded due to low fit, leaving the final form with 43 items. The Cronbach's alpha coefficients were .83 for OSIVQ, .82 for the object-imagery subscale, .85 for the spatial-imagery subscale, and .77 for the verbal subscale. In Study 1, OSIVQ was used to assess the convergent and divergent validity of IVIVTQ subscales. The internal consistency coefficient for the object-imagery, the spatial-imagery, and the verbal subscales of OSIVQ were .87, .84, and .83, respectively, in current study.

2.1.2.8 Depression Anxiety Stress Scale

Lovibond and Lovibond (1995) developed a 21-item version of DASS by selecting items from the long version of Depression Anxiety Stress Scale. Each subscale of DASS-21 consists of 7 items. The items are rated on a 4-point Likert scale, ranging from 0 (did not apply to me at all) to 7 (applied to me very much or most of the time). Higher scores from each subscale indicate higher severity of symptoms. Sample items include ''I was worried about situations in which I might panic and make a fool of myself'', ''I was unable to become enthusiastic about anything," and "I felt that I was rather touchy".

Yıldırım et al. (2018) adapted DASS-21 to Turkish. Confirmatory factor analysis yielded that the tripartite model comprising depression, anxiety, and stress factors proposed by Willemsen et al. (2011) showed the best fit for data. The Cronbach's alpha coefficients of each subscale were .89 (depression), .87 (anxiety), and .90 (stress). The test-retest reliability analysis showed that correlations between baseline scores and two weeks follow-up scores were .93 (depression), .83 (anxiety), and .82 (stress). In terms of convergent validity, each subscale of DASS-21 had high significant correlations with the Beck Depression Inventory (r = .78, r = .74, and r = 71; respectively), Beck Anxiety Inventory (r = .73, r = .87, and r = 77; respectively), Dissociative Experiences Scale (r = .57, r = .61, and r = 61; respectively), Somatoform Dissociation Questionnaire *(*r = .55, r = .65, and r = 58; respectively), and Toronto Alexithymia Scale (r = .61, r = .60, and r = 61; respectively), supporting convergent validity of the Turkish form. Furthermore, DASS-21 subscales were able to distinguish between depressive patients, anxious patients, and non-clinical

participants (Yıldırım et al., 2018). In Study 1, DASS-21 was used to assess the incremental validity of PSAS and the convergent validity of IVIVTQ subscales. The internal consistency coefficient for depression, anxiety, and stress subscales of DASS were .89, .83, and .88, respectively, in current study.

1.1.2.9 Gender Role Attitudes Scale

Gender Role Attitudes Scale (GRAS) was originally developed by García-Cueto and colleagues (2015) to assess gender role attitudes from the perspective of gender equality. The scale consists of 20 items rated on 5-point Likert scale, ranging from 1 (strongly disagree) to 5 (strongly agree). Higher scores refer to the higher endorsement of gender equality. Sample items read as ‘‘Household chores should not be allocated by sex’’ and ‘‘I think it is right that in my circles of friends, my future domestic activity is considered more important than my professional activity”. The scale has a one-factor structure. The Turkish adaptation of the scale was made by Bakioğlu and Türküm (2019). Cronbach’s alpha coefficient was .88. The test-retest analysis indicated that the correlation between baseline and 15-day follow-up scores was .77. Both exploratory and confirmatory factor analyses yielded a one-factor structure similar to the original scale. In terms of convergent validity, the Turkish form of GRAS had negative correlations with the Benevolent Sexism (r = -.42), Hostile Sexism (r = -.53), Heterosexual Intimacy (r = -.36), Protective Paternalism (r = -.42), and Complementary Gender Differentiation (r = -.19) subscales of Ambivalent Sexism Inventory. In Study 1, the scale was used to assess the divergent validity of PSAS and IVIVTQ. The internal consistency coefficient for GRAS was .89 in the current study.

2.1.3 Procedure

The developers of the PSAS and IVIVTQ were contacted via e-mail for their permission regarding the adaption of the scales into Turkish. After the authors’ consent, the ethical approval for the first study was obtained from the Human Subjects Ethics Committee of the Middle East Technical University. The scale adaptation procedure was conducted according to the guidelines of the International

Test Commission (Hernández et al., 2020). A double-translation and reconciliation procedure was applied (International Test Commission, 2017). The author of the thesis and a senior psychology researcher translated the scales into Turkish, and a bilingual (Turkish-English) scholar combined the translations into single forms. Then pilot studies with 25 people were carried out to assess the clarity of the translations. According to the feedback from the pilot studies, the final versions of the Turkish forms were formed. The final versions of two scales and other scales of the first study were presented online to participants in counter-balanced order via Qualtrics Survey system. All participants were informed about the purpose of the study, confidentiality procedures, and their right to refuse or quit the survey at any time during the study process. It took an average of 20 minutes to complete the online questionnaire package.

2.1.4 Statistical Analyses

The data was analyzed using SPSS 25 and JASP 0.16.1. Exploratory factor analyses (EFA) were conducted according to recommendations by Brown (2015), Costello and Osborne (2005), and Watkins (2020). The assumptions of linearity, univariate, and multivariate normality were assessed by checking scatter plots, skewness (< 2.0) and kurtosis (< 7.0) values, and Mardia's test, respectively. Criteria of item correlations between .30 and .80, Kaiser-Meyer-Olkin (KMO) test of sampling (KMO value over .70), and Bartlett's test of sphericity were utilized to assess the suitability of data for EFA (Brown, 2015; Watkins, 2020). Sample sizes were above 130, which was the minimum recommended number of participants based on low communality, variable/factor ratio of 8, and six-factor structure (Mundfrom et al., 2005). The number of factors was decided on the results of parallel analysis, scree plot, Kaiser's criterion of eigenvalue equal or greater than 1.0, and theory (Brown, 2015; Costello & Osborne, 2005; Watkins, 2020). A loading of .40 was used as the threshold loading of an item to a factor.

Confirmatory factor analyses (CFA) were conducted in accordance with the recommendations of Brown (2015) and Hair et al. (2019). The assumptions of

univariate and multivariate normality were assessed by checking skewness (< 2.0) and kurtosis (< 7.0) values and Mardia's test, respectively. Sample sizes were above 200, which was advised by both rule of thumb in literature and Monte-Carlo simulation studies (Kyriazos, 2018). CFAs were performed to test the factor structures of Turkish forms found in EFAs. The model fit was assessed with the chi-square test, standardized root-mean-square residual (SRMR), root-mean-square error of approximation (RMSEA), comparative fit index (CFI), and Tucker-Lewis index (TLI). An insignificant χ^2 test (a significant test is expected when $N > 250$ and the number of observed variables is more than 12), SRMR values ≤ .08, RMSEA values ≤ .10, TLI and CFI values > .90 suggest adequate model fit (Brown, 2015; Hair et al., 2019; Hu & Bentler, 1999; Schermelleh-Engel et al., 2003).

The reliabilities of Turkish forms were examined with internal consistency coefficients and the test-retest reliability method in the subsample. Cronbach's alpha values over .70-.80 (Hoekstra et al., 2019) and test-retest correlations over .75 (Matheson, 2019) imply good reliability. The sample size for test-retest reliability was over the recommended sample size of 66, which is required to detect a minimum inter-class correlation of .30 at a power of .80 (Bujang & Baharum, 2017). Convergent-divergent, incremental, and discriminant validities were assessed by Pearson correlation coefficients, multiple regression analysis, and independent t-tests in the subsample, respectively. The assumptions of regression were checked by scatter plot, Q-Q plot standardized residuals, Durbin-Watson value of 2.00, residuals versus predicted plot, and tolerance (> .01) and VIF (< 10) values. Finally, the homogeneity of variance assumption of the independent t-test was tested by Levene's test.

2.2. RESULTS

2.2.1 Psychometric Properties of PSAS

2.2.1.1 Exploratory Factor Analysis

An EFA was conducted to analyze the underlying factor structure of PSAS in the first sample. Multiple outliers were detected using Mahalanobis distance. Four outliers were removed from further analyses. The final sample consisted of 322 participants. Data were screened for linearity, univariate and multivariate normality, and the assumption of multivariate normality (Mardia*'s* $Z = 12.80$, $p < .001$) was not met.

Most of the correlations between the items of PSAS were between .3 and .8 (Mayers, 2013). Overall, the Kaiser-Meyer-Olkin's (KMO) test of sampling adequacy was .91, and Bartlett's test of sphericity was significant ($\chi^2(120) = 3065.18$, $p < .001$). These results indicated that the data was suitable for EFA. Due to expected factor correlation and violation of multivariate normality, a parallel analysis with principal axis factoring estimation and oblique-oblimin rotation was conducted. Parallel analysis, Kaiser's criterion of eigenvalue, scree plot examination, and theory suggested a two-factor structure (see Figure 3 for Scree plot). All items except Item 8 (Being distracted by sounds, noise in the environment. (e.g., ticking of the clock, house noises, traffic) had loadings greater than .40. Item 8 was removed from further analyses, and a second solution was tested. The final solution with a two-factor structure explained 53.90% of the total variance. The cognitive arousal factor explained 30.70% of the total variance, while the somatic arousal factor explained 23.10% of the variance. All items had loadings within a range between .45-.95. The factor loadings of the final solution can be seen in Table 4. Factor 1 included seven items measuring cognitive PSA, while Factor 2 comprised eight items assessing somatic PSA. The results showed that Turkish PSAS had the same factor structure found in the original study.

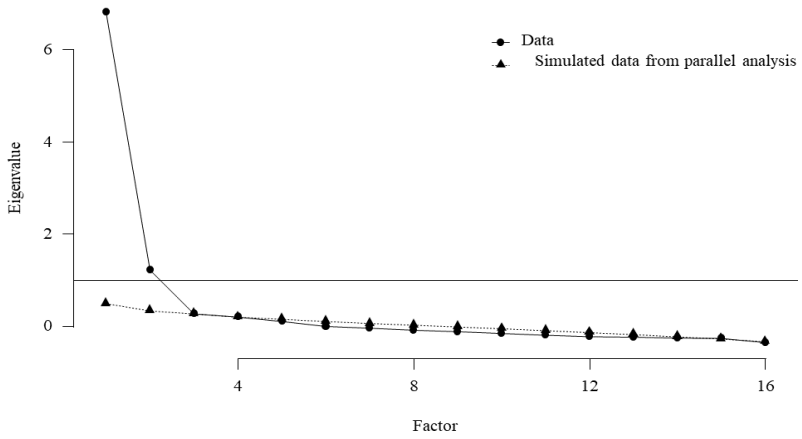


Figure 3 Initial Scree Plot of The Factor Structure of PSAS

Table 4 Factor Structure of PSAS

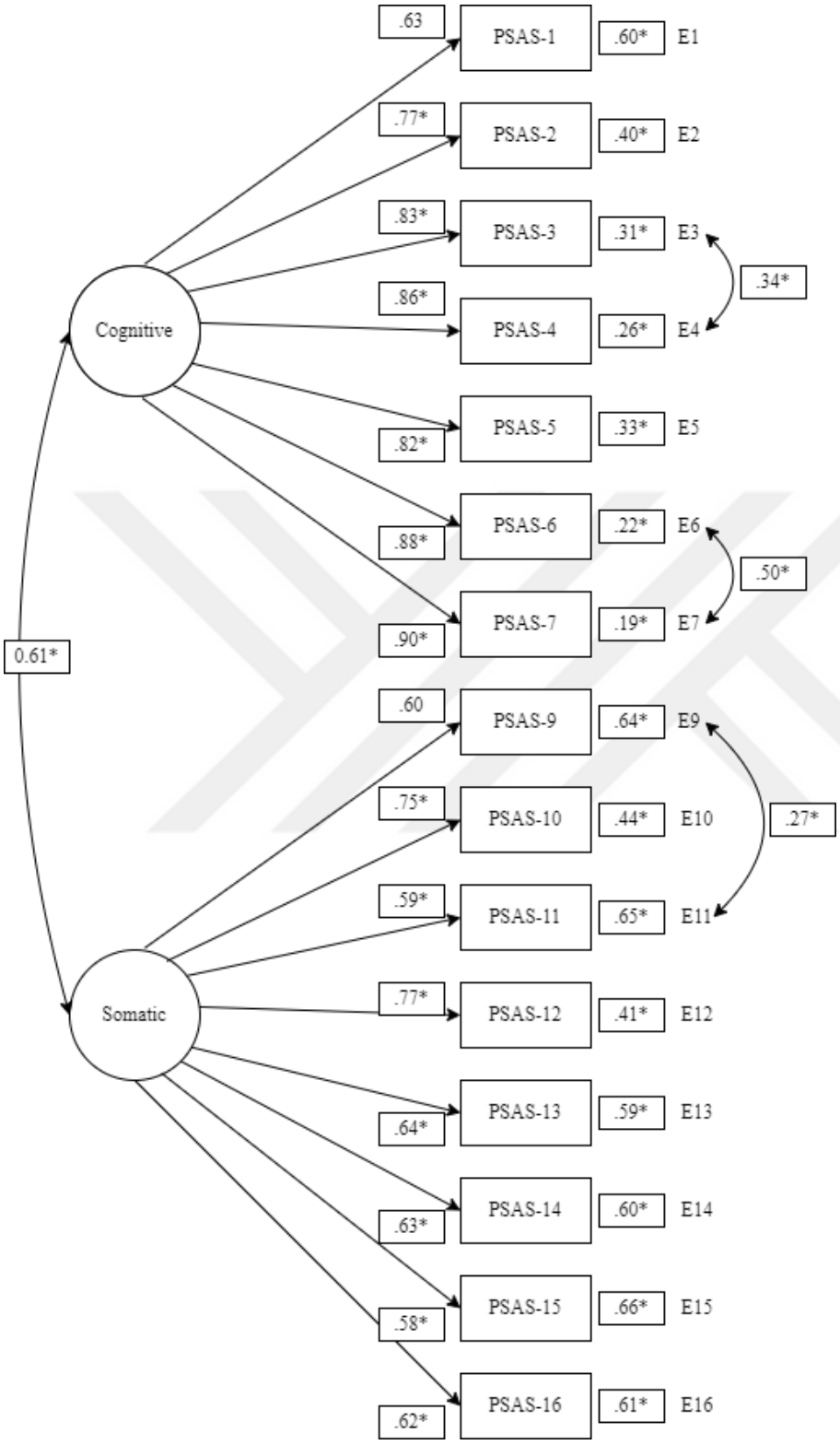
	Factor	
	Cognitive	Somatic
1. Worry about falling asleep.	**.48**	
2. Review or ponder the events of the day.	**.77**	
3. Depressing or anxious thoughts.	**.70**	
4. Worry about problems other than sleep.	**.86**	
5. Being mentally alert, active.	**.72**	
6. Can't shut off your thoughts.	**.91**	
7. Thoughts keep running through your head.	**.95**	
9. Heart racing, pounding or beating irregularly.		**.74**
10. A jittery, nervous feeling in your body.		**.61**
11. Shortness of breath or labored breathing.		**.69**
12. A tight, tense feeling in your muscles.		**.76**
13. Cold feeling in your hands, feet or your body in general.		**.45**
14. Have stomach upset (knot or nervous feeling in stomach, heartburn, nausea, gas, etc.).		**.61**
15. Perspiration in palms of your hands or other parts of your body.		**.50**
16. Dry feeling in mouth or throat.		**.61**

Note. Bold characters show items loaded to factors in respective columns.

2.2.1.2 Confirmatory Factor Analysis

To identify the latent factor structure of PSAS, a CFA was conducted in the second sample. There were no missing values in the data. Multiple outliers were detected using Mahalanobis distance. Eleven outliers were removed from further analyses. The final sample consisted of 314 participants. The data was screened for univariate and multivariate normality, adequate sample size was achieved, and continuous scale measurement and all assumptions were met, except multivariate normality (Mardia*'s* $Z = 10.36$ $p < .001$).

A two-factor model of PSAS with 15 items was hypothesized based on EFA results. Seven items were entered as the indicators of PSAS-C, and the remaining eight items were entered as the indicators of PSAS-S. Due to the violation of multivariate normality, CFA was carried out using the maximum likelihood parameter estimates with Satorra-Bentler correction and robust error calculation. The overall goodness-of-fit indices revealed that the two-factor model did not fit the data well ($\chi^2(89) = 365.89$, $p < .001$, SRMR = .06, RMSEA = 0.10, *90% CI* = [.09,.11], TLI = .89, CFI = .91). To improve the initial model, several error covariances were added to the model as suggested by modification indices (items 3-4, items 6-7, and items 9-11). Each modification significantly contributed to the model fit. The final model fit the data adequately ($\chi^2(86) = 257.63$, $p < .001$, SRMR = .06, RMSEA = .08, *90% CI* [.07,.09], TLI = .93, CFI = .94). Figure 4 depicts the path diagram of the Turkish version of PSAS. The results confirmed the factor structure suggested in the original study. The following analyses were conducted with the two-factor structure of PSAS with 15 items.



Note. * $p < .001$. All reported estimates are standardized.

Figure 4 Path Diagram of The Two-Factor Model of Turkish PSAS

2.2.1.3 Convergent and Divergent Validity

The convergent and divergent validities were tested by examining Pearson correlations between PSAS, PSAS-C, PSAS-S, and the somatic subscale of BAI, RTSQ, ISI, and GRAS in the subsample. Multiple outliers were detected using Mahalanobis distance. No multiple outliers were found. The final sample consisted of 556 participants.

It was expected that there would be significant positive correlations between PSAS-C and RTSQ, between PSAS-S and the somatic subscale of BAI, and between PSAS and ISI, while PSAS-C and PSAS-S would not be significantly correlated with GRAS. The correlations between the scales can be seen in Table 5. The results indicated that there was a positive correlation between PSAS-C and RTSQ (r = .72, p < .001), between PSAS-S and the somatic subscale of BAI (r = .67, p < .001), and between PSAS and ISI (r = .65, p < .001). In addition, it was found that GRAS had significant but small positive correlations with PSAS (r = .25, p < .001), PSAS-C (r = .28, p < .001), and PSAS-S (r = .15, p < .05). The results suggested that Turkish form of PSAS has good convergent and acceptable divergent validity.

Table 5 The correlations among PSAS, PSAS-C, PSAS-S, BAI-Somatic, RTSQ, ISI, and GRAS

Scales	1	2	3	4	5	6	7	8	9
1. PSAS	-								
2. PSAS-C	.91**	-							
3. PSAS-S	.88**	.60**	-						
4. RTSQ	.67**	.72**	.46**	-					
5. BAI-Somatic	.61**	.44**	.67**	.38**	-				
6. GRAS	.25**	.28**	.15**	.26**	.11*	-			
7. ISI-1	.59**	.60**	.43**	.38**	.33**	.12*	-		
8. ISI-2	.41**	.35**	.39**	.20**	.31**	-.03	.53**	-	
9. ISI-3	.24**	.17**	.26**	.08	.21**	-.08	.20**	.48**	-
10. ISI	.65**	.62**	.54**	.42**	.42**	.08	.79**	.68**	.46**

Note 1. * p < .01, ** p < .001

Note 2. PSAS: Pre-sleep Arousal Scale, PSAS-C: Pre-sleep Arousal Scale - Cognitive Subscale, PSAS-S: Pre-sleep Arousal Scale - Somatic Subscale, RTSQ: Ruminative Thought Style Questionnaire, BAI-Somatic: Beck Anxiety Inventory - Somatic Subscale, GRAS: Gender Role Attitude Scale, ISI: Insomnia Severity Index, ISI-1: Difficulty falling asleep, ISI-2: Difficulty staying asleep, ISI-3: Waking up too early.

2.2.1.4 Incremental Validity

To assess the incremental validity of PSAS, a two-step hierarchical multiple regression was conducted to predict insomnia severity, using cognitive and somatic PSAs, depression, anxiety, and stress as the predictors in the subsample. Multiple outliers were detected using Mahalanobis distance. One outlier was removed from further analyses. The final sample consisted of 555 participants. The assumptions of linearity, normality of residuals, independence of observations, homoscedasticity, and multicollinearity were met.

Depression, anxiety, and stress were entered in step one, and cognitive and somatic PSAs were entered in step two. The first model significantly predicted insomnia severity, F (3,551) = 62.67, $p < .001$, $R^2 = .25$. Adding cognitive and somatic PSAs to the regression model explained an additional 17.70% of the variance in insomnia severity, F (5,549) = 83.18, $p < .001$, and depression and anxiety became insignificant, the total explained variance increased to 42.60%. Regression coefficients and standard errors can be found in Table 6. These results support the incremental validity of cognitive and somatic PSAs in explaining additional variance in insomnia severity.

Table 6 Hierarchical Regression Analysis Summary for Depression, Anxiety, Stress, and Pre-Sleep Arousal Predicting Insomnia Severity

						Collinearity Statistics	
Model	*B*	*SE*	*B*	*t*	*p*	Tolerance	VIF
Step 1 Intercept	4.78	.39		12.25	< .001		
Depression	.18	.06	.17	3.23	.001	0.47	2.15
Anxiety	.37	.07	.29	5.00	<.001	0.40	2.50
Stress	.10	.07	.08	1.48	.14	0.34	2.98
Step 2 Intercept	-1.19	.59		-2.04	.04		

Table 6 (continued)

Depression	.04	.05	.04	.81	.42	0.44	2.27
Anxiety	.07	.08	.06	.96	.34	0.28	3.62
Stress	-.03	.06	-.03	-.45	.65	0.32	3.11
Cognitive Arousal	.32	.03	.45	10.13	< .001	0.54	1.87
Somatic Arousal	.19	.04	.23	4.37	< .001	0.39	2.56

2.2.1.5 Discriminant Validity

To investigate the discriminant validity of the Turkish PSAS, it was tested whether there were significant differences between scores of normal sleepers and insomniacs on PSAS subscales. Data were screened for normality and homogeneity of variances. The assumption of univariate normality was met for both PSAS-C and PSAS-S, but the assumption of homogeneity of variances was violated for PSAS-S. The analysis was conducted with the subsample (N = 556).

ISI cut-off score of 8 (Bastien et al., 2001) was used to differentiate normal sleepers from insomniacs. Two independent samples t-tests were conducted. There was a significant difference in PSAS-C scores of normal sleepers (M = 16.52, SD = 6.32, N = 239) and insomniac groups (M = 23.65, SD = 6.29, N = 317); t (554) = -13.21, p < .001, d = -1.13 (see Figure 5). Due to the violation of the equality of variances assumption, a Welch t-test was conducted for PSAS-S score. The results showed that there was a significant difference in PSAS-S scores of normal sleepers (M = 12.62, SD = 4.69, N = 239) and insomniac groups (M = 17.79, SD = 6.32, N = 317); t (554) = -10.63, p < .001, d = -.91 (See Figure 6). The result yielded that Turkish form of PSAS had good discriminant validity.

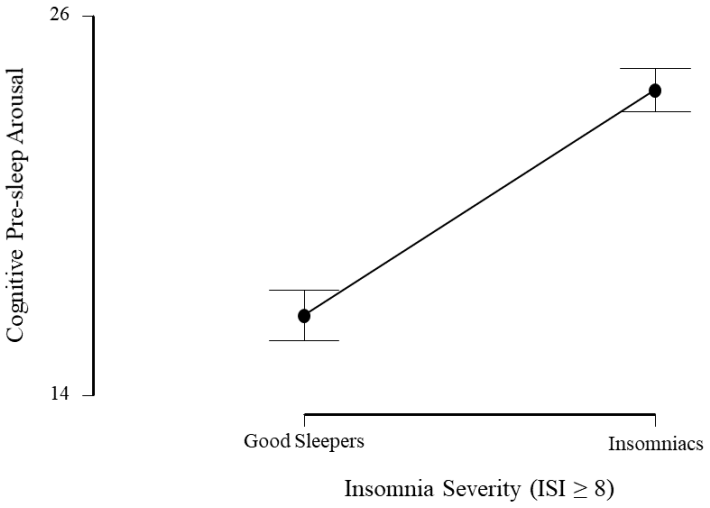


Figure 5 Pre-Sleep Cognitive Arousal Scores as a Function of High and Low Insomnia Severity

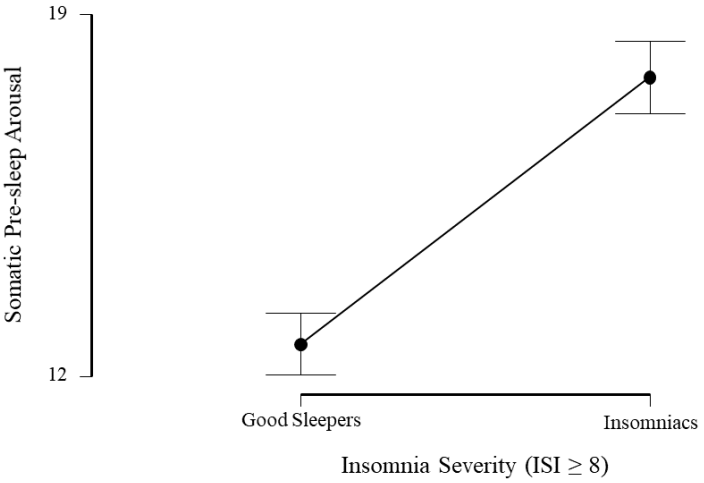


Figure 6 Pre-Sleep Somatic Arousal Scores as a Function of High and Low Insomnia Severity

2.2.1.6 Reliability

The reliability analysis in the subsample (N = 556) showed that Cronbach's alpha internal consistency values for PSAS, PSAS-C, and PSAS-S were .92, .93, and .86, respectively. Three-week test-retest reliability of PSAS was tested in the third sample of 88 participants (80.68% female). The intra-class correlation coefficients between the scores were .90 (p < .001), .90 (p < .001), and .83 (p < .001) for PSAS, PSAS-C, and PSAS-S respectively. The results revealed that the Turkish form had good reliability.

2.2.2 Psychometric Properties of IVIVTQ

2.2.2.1 Exploratory Factor Analysis

The underlying factor structure of IVIVTQ was analyzed with the first sample, using EFA. Multiple outliers were detected using Mahalanobis distance. Nine outliers were removed from further analyses. The final sample consisted of 317 participants. Data were screened for linearity, univariate and multivariate normality, and the assumption of multivariate normality (Mardia's Z = 15.73, p < .001) was not met.

Most of the correlations between items of IVIVTQ were higher than .3 and lower than .8. Overall, the KMO test of sampling adequacy was .94, and Bartlett's tests of sphericity were significant ($\chi^2(190) = 5428.02$, p < .001), meaning that the sample was suitable for EFA. Due to the violation of multivariate normality, a parallel analysis with principal axis factoring and oblique-oblimin rotation was conducted. Parallel analysis and Kaiser's criterion indicated a three-factor structure, while scree plot examination and the theory suggested two factors (see Figure 7 for Scree plot). Therefore, a second solution with two factors was tested. The final solution yielded a two-factor structure that explained 61.40% of the variance. IVI subscale explained 33.30% of the total variance, while the IVT subscale explained 28.10% of the variance. There were no cross-loading items, and all items had loadings greater than .40 within a range between .42-.92. The factor loadings for IVIVTQ can be seen in Tables 8. Factor 1 included ten items related to IVI, while Factor 2 had ten items

pertaining to IVT. The results showed that Turkish IVIVTQ had the same factor structure found in the original study.

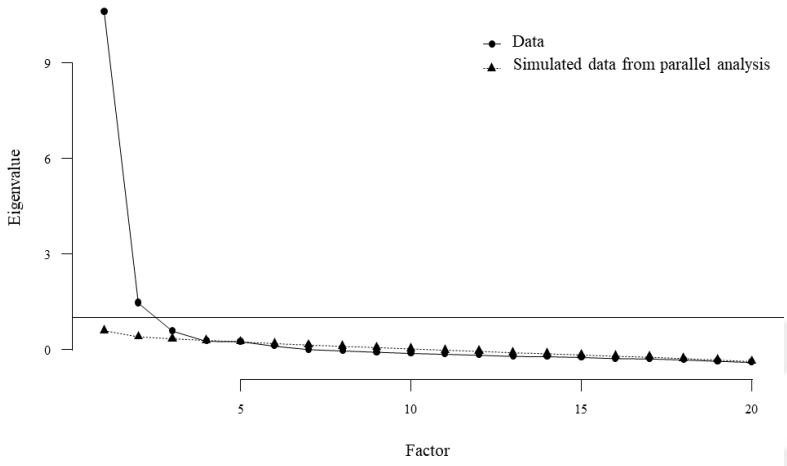


Figure 7 Initial Scree Plot of The Factor Structure of IVIVTQ

Table 7 Factor Structure of IVIVTQ

	Factor	
	Intrusive Visual Imagery	Intrusive Verbal Thoughts
1. There are images that come to mind that I cannot erase.	**.78**	
2. My thoughts frequently return to one image.	**.71**	
3. I have images in my mind that I cannot stop.	**.91**	
4. There are images that keep jumping into my head.	**.75**	
5. I find it hard to sleep as images keep coming into my head.	**.50**	
6. There are negative images from my past that keep coming to mind.	**.81**	
7. When I have had an argument with someone, I will keep seeing images from it in my mind's eye for the next few days, even though I do not want to.	**.44**	
8. I often picture images of things that will happen in the future, without meaning to.	**.42**	

Table 7 (continued)

9. There are some images that enter my head without me being able to avoid it.	**.88**	
10. I keep seeing events from my past in my mind's eye, against my will.	**.76**	
11. There are verbal thoughts that come to mind that I cannot erase.		**.89**
12. My thoughts frequently return to one word or phrase.		**.81**
13. I have verbal thoughts in my mind that I cannot stop.		**.92**
14. There are verbal thoughts that keep jumping into my head.		**.83**
15. I find it hard to sleep as verbal thoughts keep coming into my head.		**.76**
16. There are negative verbal thoughts about my past that keep coming to mind.		**.67**
17. When I have had an argument with someone, I will keep having verbal thoughts about it for the next few days, even though I do not want to.		**.72**
18. I often have verbal thoughts about things that will happen in the future, without meaning to.		**.66**
19. There are some words or phrases that enter my head without me being able to avoid it.		**.77**
20. I keep hearing word or phrases from my past in my head, against my will.		**.69**

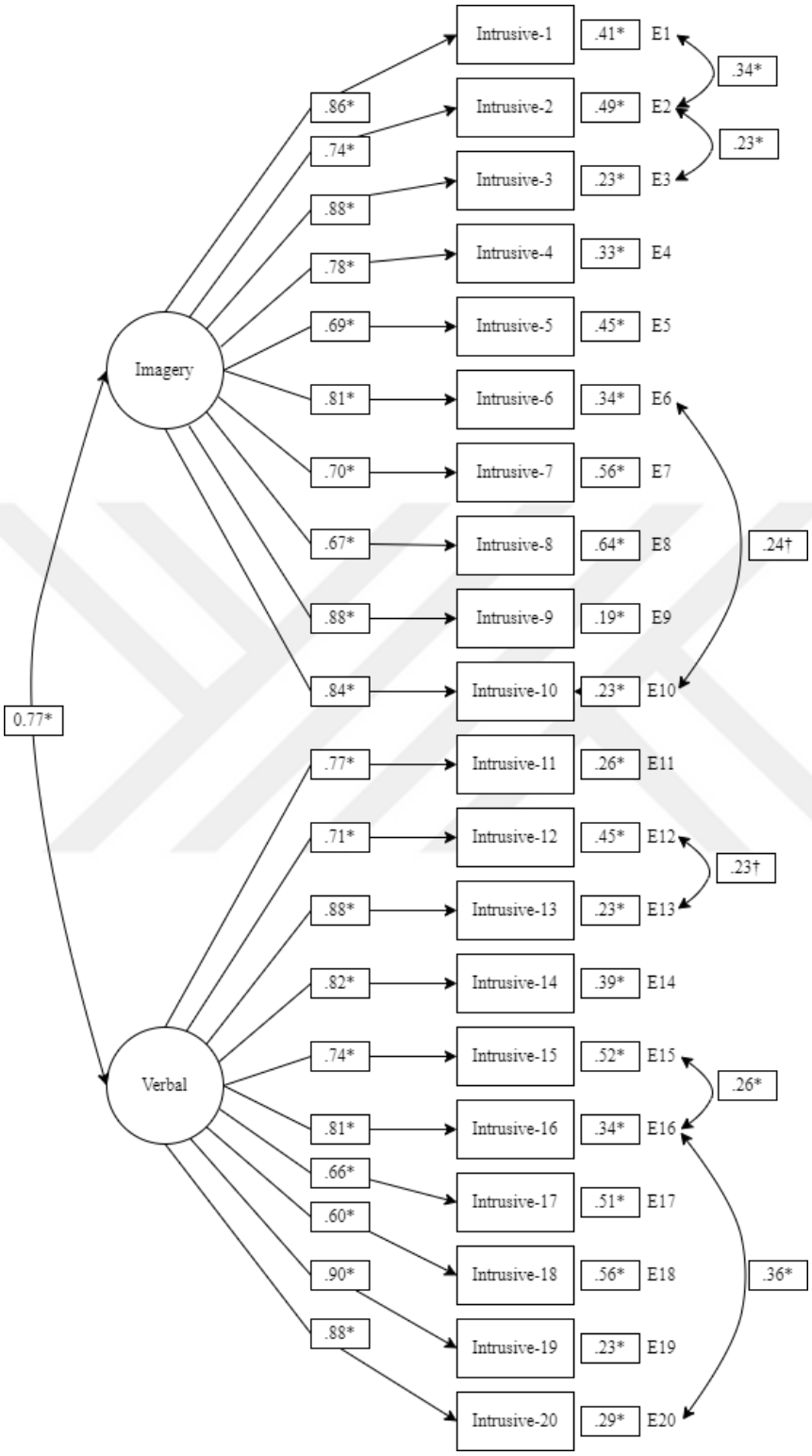
Note. Bold characters show items loaded to factors in respective columns.

2.2.2.2 Confirmatory Factor Analysis

The latent factor structure of IVIVTQ in the Turkish sample was identified using the second sample with CFA. There were no missing values in the data. Multiple outliers were detected using Mahalanobis distance. Twelve outliers were removed from further analyses. The final sample consisted of 313 participants. Before analysis, data were screened for univariate and multivariate normality, adequate sample size, and continuous scale measurement, and all assumptions, except multivariate normality (Mardia's $Z = 18.43$, $p < .001$), were met.

According to EFA results, a two-factor model of IVIVTQ with 20 items was hypothesized. The model had two factors, intrusive visual imagery and verbal thought, each having 10-items. Due to the violation of multivariate normality, CFA

was carried out using the maximum likelihood parameter estimates with Satorra-Bentler correction and robust error calculation. The overall goodness-of-fit indices suggested that 2-factor model did not fit the data well ($\chi^2(169) = 753.45$, $p < .001$, SRMR = .05, RMSEA = 0.11, *90% CI* = [.10,.11], TLI = .88, CFI = .89). To improve the initial model, several error covariances were added to the models as suggested by modification indices (items 1-2, items 2-3, items 6-10, items 12-13, items 15-16, items 16-20). Each modification significantly contributed to the model fit. The final two-factor model had acceptable fit ($\chi^2(163) = 612.06$, $p < .001$, SRMR = .05, RMSEA = .09, *90% CI* [.09,.10]; TLI = .90; CFI = .92). Figure 8 depicts path diagrams of the Turkish version of IVIVTQ. The results confirmed the factor structure found in the original study. The following analyses were conducted with the two-factor structure of IVIVTQ.



Note. † < .01, *p < .001. All reported estimates are standardized.

Figure 8 Path Diagram of The Two-Factor Model of The Turkish IVIVTQ Form

2.2.2.3 Convergent and Divergent Validity of IVIVTQ

The convergent and divergent validities were assessed by examining Pearson correlations between IVIVTQ, OSIVQ, DASS, and GRAS using the subsample. Multiple outliers were detected using Mahalanobis distance. One outlier was removed from further analyses. The final sample consisted of 554 participants.

It was expected that there would be significant positive correlations between IVIVTQ-Visual and the object imagery subscale of OSIVQ (OSIVQ-Object) and the subscales of DASS. Similarly, it was expected that there would be significant positive correlations between IVIVTQ-Verbal and the subscales of DASS. On the other hand, it was expected that the subscales of IVIVTQ would not be significantly correlated with GRAS. The correlations between the scales can be seen in Table 9. The results revealed that there was a moderate positive correlation only between IVIVTQ-Visual and OSIVQ-Object ($r = .37$, $p < .001$). In addition, there was a small but significant correlation between IVIVTQ-Verbal and OSIVQ-Object ($r = .17$, $p < .001$). As expected, IVIVTQ and its subscales were significantly associated with the subscales of DASS. In contrast to the hypothesis, GRAS had small positive correlations with IVIVTQ-Visual ($r = .21$, $p < .001$) and the IVIVTQ-Verbal ($r = .22$, $p < .001$). The results showed that Turkish IVIVTQ had good convergent and acceptable divergent validities.

Table 8 The correlations among IVIVTQ, OSIVQ, DASS, and GRAS

Scales	1	2	3	4	5	6	7	8	9
1. IVIVTQ	-								
2. IVIVTQ-Visual	.93***	-							
3. IVIVTQ-Verbal	.93***	.74***	-						
4. OSIVQ-Object	.29***	.37***	.17**	-					
5. OSIVQ-Spatial	-.08*	-.04	-.12**	.19***	-				
6. OSIVQ-Verbal	-.04	-.02	.05	.20***	-.18***	-			
7. DASS-D	.50***	.44***	.50***	.10*	-.05	-.06	-		
8. DASS-A	.54***	.49***	.52***	.17***	-.05	-.03	.65***	-	
9. DASS-S	.55***	.50***	.52***	.14**	-.12**	-.01	.71***	.76***	-
10. GRAS	.23***	.21**	.22***	.17***	-.24***	.17***	.20***	.17***	.24***

Note 1. * $p < .05$, ** $p < .01$, *** $p < .001$

Note 2. IVIVTQ: Intrusive Visual Imagery and Verbal Thought Questionnaire, IVIVTQ-Visual: Intrusive Visual Imagery Subscale of IVIVTQ, IVIVTQ-Verbal: Intrusive Verbal Thought Subscale of IVIVTQ, OSIVQ – Object: Object - Spatial Imagery and Verbal Questionnaire Object Imagery Subscale, OSIVQ – Spatial: Object - Spatial Imagery and Verbal Questionnaire Spatial Imagery Subscale, OSIVQ – Verbal: Object - Spatial Imagery and Verbal Questionnaire Verbal Subscale, DASS-D: Depression Anxiety Stress Scale - Depression Subscale, DASS-A: Depression Anxiety Stress Scale - Anxiety Subscale, DASS-S: Depression Anxiety Stress Scale Stress - Subscale, GRAS: Gender Role Attitude Scale.

2.2.2.4 Reliability of IVIVTQ

Cronbach's alpha internal consistency values for IVIVTQ, IVIVTQ-Visual, and IVIVTQ-Verbal were found as .96, .93, and .94 in the subsample (N = 556), respectively. In a sample of 88 participants (80.68% female), three weeks of test-retest reliability of IVIVTQ was examined. The intra-class correlation coefficients between the scores were .91 ($p < .001$), .83 ($p < .001$), and .91 ($p < .001$) for IVIVTQ, IVTQ-Visual, and IVIVTQ-Verbal, respectively. The results revealed that the Turkish form of IVIVTQ had good reliability.

CHAPTER 3

STUDY 2: THE MODEL TESTING

METHOD

3.1.1 Participants

Data for the second study was collected from 1444 participants. The participants were recruited via social media and the METU Psychology department's online data collection system, SONA. To determine the individuals with primary insomnia disorder, DSM-V Insomnia Disorder criteria (American Psychiatric Association, 2013) and ISI scores over eight were utilized (Bastien et al., 2001). The participants were included in the analysis if they reported having: (1) sleep problems (difficulty falling asleep, disturbed sleep, or early morning awakenings); (2) significant distress and impairment due to sleep problems; (3) sleep problems three and more days in a week; (4) insomnia symptoms more than three months; (5) sleep difficulties despite having the adequate opportunity for sleep; (6) no sleep-wake disorders; (7) no secondary insomnia related to mental, medical, and neurological disorders; (8) no secondary insomnia related to the substance or medication use. The list of disorders, substances, and medications that may cause secondary insomnia was derived from the guidelines of Schutte-Rodin Sharon et al. (2008) and Rieman et al. (2017). The exclusion list can be seen in Appendix N. Finally, 168 participants met the criteria for primary insomnia disorder. The exclusion procedure can be seen in Figure 9.

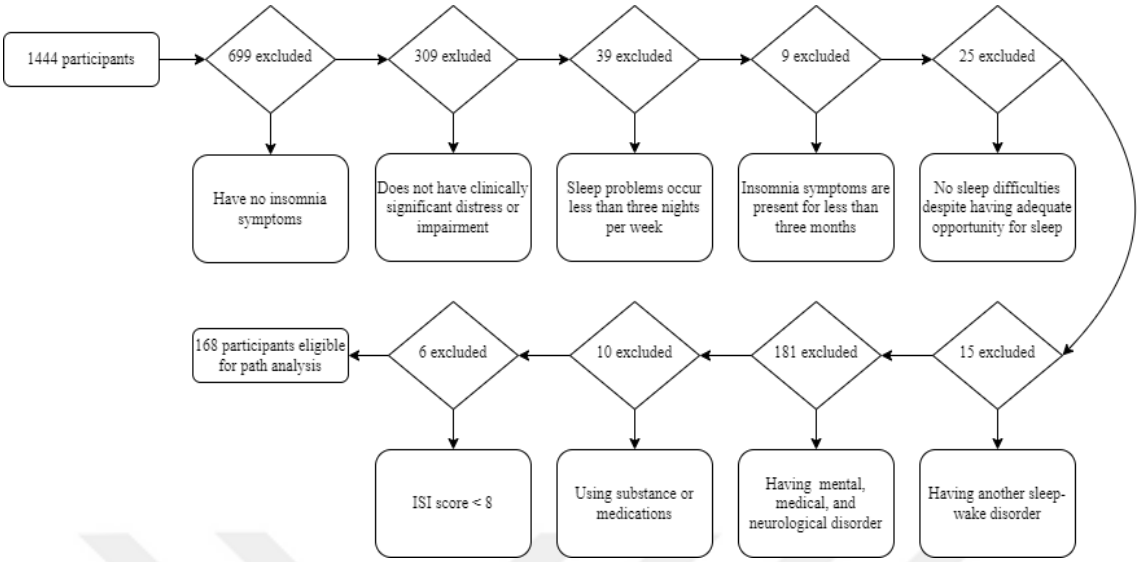


Figure 9 The Exclusion Chart

The mean age of the participants was 25.55 (*SD* = 5.26). There were 126 females (75.00%) and 42 (25.00%) males in the sample. Half of the participants had university-level education. Only 35.12% of the participants were working. The majority of the participants (88.69%) were single. Finally, 47.62% of the sample had middle socio-economic status. The demographic characteristics of the participants can be seen in Table 10.

Table 9 Demographics and Sleep Descriptives of The Sample

	M	*SD*
Age	25.55	5.26
Frequency of insomnia symptoms (days in a week)	5.16	1.52
Duration of insomnia symptoms (years)	4.11	3.54
	N	%
Gender		
Female	126	75.00
Male	42	25.00
Education		
University	84	50.00
Graduate	84	50.00
Employment		
No	109	64.88
Yes	59	35.12

Table 9 (Continued)

Marital Status		
Single	147	87.50
Married	19	11.31
Divorced	2	1.19
Socioeconomic Status		
Very Low	32	19.05
Low	46	27.38
Middle	80	47.62
High	9	5.35
Very High	1	.60
Insomnia Symptoms		
Difficulty falling asleep	150	89.29
Difficulty staying asleep	45	73.21
Waking up too early	36	21.43

3.1.2 Instruments

3.1.2.1 Demographic Information Form

The Demographic Information Form of the first study was used to assess the characteristic of the participants. Questions asking about participants' age, gender, level of education, employment status, marital status, and socioeconomic status were included. The form also had several additional questions adapted from the study of Benbir et al. (2015) regarding smoking, daily screen time, amount of consumption of black tea and coffee at the evening, exercise frequency and duration, and pre-sleep screen time. The questions can be seen in Appendix F.

3.1.2.2 Diagnostic Questions for DSM-V Insomnia Disorder

To detect primary insomnia patients, twelve questions were formulated that correspond to eight DSM-V diagnostic criteria for Insomnia Disorder. The participants were asked to report the type of their sleep complaints; the presence of significant distress and impairment associated with symptoms; the weekly frequency of their sleep difficulty; the duration of their sleep difficulty; the presence of adequate opportunity for sleep; the presence of other sleep-wake disorders, mental disorders,

medical disorders, or neurological disorders; the presence of substance abuse; and use of medication. The questions can be seen in Appendix L.

3.1.2.3 Intrusive Visual Imagery and Verbal Thought Questionnaires

Intrusive Visual Imagery and Verbal Thought Questionnaires (IVIVTQ) measure IVI and IVT. The detailed information regarding the scale can be seen in the 2.1.2.3 Intrusive Visual Imagery and Verbal Thought Questionnaires section. In Study 2, the subscales of IVIVTQ were used to assess the intrusive thoughts of the participants. The internal consistency coefficient for the intrusive visual imagery and verbal thoughts subscales of IVIVTQ were .94 and .93, respectively, in current study.

3.1.2.4 Pre-sleep Arousal Scale

Pre-sleep Arousal Scale (PSAS) measures cognitive and somatic PSA. The detailed information regarding the scale can be seen in the 2.1.2.2 Pre-sleep Arousal Scale section. In Study 2, PSAS was used to assess the PSA of the participants. The internal consistency coefficient for PSAS was .88 in current study.

3.1.2.5 The Object - Spatial Imagery and Verbal Questionnaire

The Object-Spatial Imagery and Verbal Questionnaire (OSIVQ) measures object imagery, spatial imagery, and verbal cognitive styles. The detailed information regarding the scale can be seen in 2.1.2.7 The Object - Spatial Imagery and Verbal Questionnaire section. In Study 2, the visual subscale of OSIVQ was used to assess the VIA of the participants. The internal consistency coefficient for the visual subscale of OSIVQ was .91 in current study.

3.1.2.6 Insomnia Severity Index

Insomnia Severity Index (ISI) measures the severity of insomnia. The information regarding the scale can be seen in the 2.1.2.6 Insomnia Severity Index section. In Study 2, ISI was used to assess the insomnia severity of the participants. The internal consistency coefficient for ISI was .66 in current study.

3.1.3 Procedure

The ethical approval for the second study was obtained from the Human Subjects Ethics Committee of the Middle East Technical University. The questionnaire package was presented online to participants in counter-balanced order using the Qualtrics Survey system. All participants were informed about the purpose of the study, confidentiality procedures, and their right to refuse or quit the survey at any time during the study process. Completing the online questionnaire package took approximately 25 minutes.

3.1.4 Statistical Analysis

The data analysis was carried out using SPSS 24.0 and JASP 0.16.1. There were no missing values in the data. Assumptions of linearity, univariate and multivariate normality, homoscedasticity, and multicollinearity were assessed by checking scatter plots, skewness (< 2.0) and kurtosis (< 7.0) values, and Mardia's test, Residuals vs. predicted plot, and tolerance (> .01) and VIF (< 10) values, respectively. Sample sizes were above the recommended size of 160, which was equal to 20 cases for every eight parameters estimated in the path models (Kline, 2015).

A hypothesized path model was constructed using IVI and IVT as the exogenous predictor variables and PSA and insomnia severity as the endogenous variables. The moderation analysis was conducted using PROCESS Macro in SPSS (Hayes, 2022). An insignificant χ^2 test (a significant test is expected when $N > 250$ and the number of observed variables are more than 12), SRMR values ≤ .08, RMSEA value ≤ .10, and TLI and CFI values > .90 indicate an adequate model fit (Brown, 2015; Hair et al., 2019; Hu & Bentler, 1999; Schermelleh-Engel et al., 2003). Mediational roles were assessed by testing the significance of indirect effects, which are effects of IVI and IVT via PSA on insomnia severity. All reported path coefficients and indirect effects were standardized.

3.2 RESULTS

3.2.1 Descriptive Statistics for The Variables of The Model

Descriptive statistics and Pearson correlations between variables can be seen in Table 11.

Multiple outliers were detected using Mahalanobis distance. Two outliers were removed from further analyses. The final sample consisted of 166 participants. PSA was significantly related to IVI (r = .48, p < .001), IVT (r = .51, p < .001), VIA (r = .21, p < .01), and insomnia severity (r = .41, p < .001). Moreover, VIA had significant correlation with IVI (r = .41, p < .001).

Table 10 The descriptive statistics and correlations among the variables of the model

Scales	1	2	3	4	*M*	*SD*	*Min*	*Max*
1. Intrusive Visual Imagery	-				33.30	9.92	10.00	50.00
2. Intrusive Verbal Thoughts	.69***	-			36.96	8.87	10.00	50.00
3. Visual Imagery Ability	.37***	.31***	-		51.60	12.52	19.00	75.00
4. Pre-sleep Arousal	.56***	.47***	.25**	-	45.27	10.34	18.00	70.00
5. Insomnia Severity	.35***	.28***	.16*	.46***	15.36	4.31	8.00	26.00

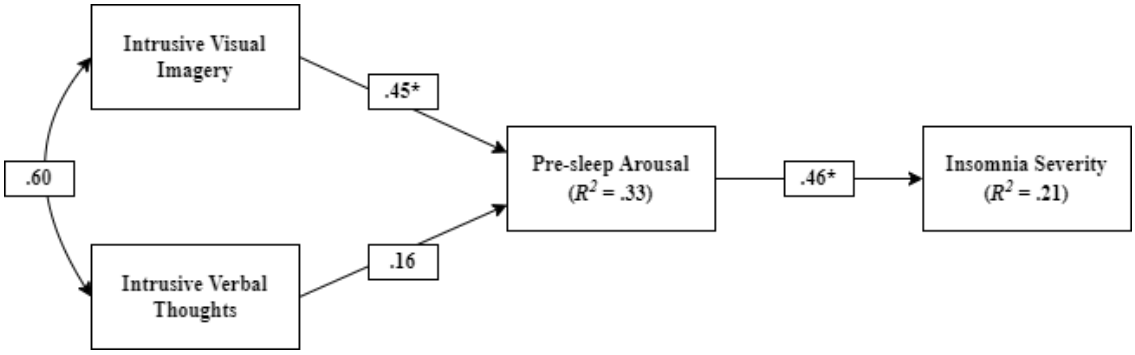
Note. * p < .05, ** p < .01, *** p < .001.

3.2.2 Path Analysis

A multivariable path model linking IVI, IVT to PSA, PSA to insomnia severity was constructed (see Figure 10). Data were screened for linearity, univariate and multivariate normality, homoscedasticity, and multicollinearity. All assumptions were met. The path model was estimated using the maximum likelihood parameter estimates with standard error calculation.

The model yielded good fit values ($\chi^2(2) = 2.76$, $p = .25$, SRMR = .03, RMSEA = 0.05, *90% CI* = [.00,.17], TLI =0.98, CFI = 0.99). The direct associations in the model showed that IVI ($\beta = .45$, $p < .001$) and IVT ($\beta = .16$, $p = .07$) significantly predicted PSA and PSA ($\beta = .46$, $p < .001$) significantly predicted insomnia severity. IVI and IVT explained 32.70% of the variance in PSA, and the total model was responsible for 21.20% of the variance in insomnia severity. Moreover, there was a statistically significant indirect effect of PSA as the mediator for IVI predicting insomnia severity (IE = .20, $p < .001$), but not for IVT predicting insomnia severity (IE = .07, $p = .08$). The results indicated that hypotheses 1, 3 and 4 were supported.

A further analysis was conducted in the path model by controlling age, gender, marital status, weekly exercise frequency, consuming black tea and coffee at the evening, smoking status, daily screen time, and pre-sleep screen time. None of the control variables had significantly predicted PSA. The direct associations in the model showed that IVI ($\beta = .40$, $p < .001$) and IVT ($\beta = .16$, $p = .07$) significantly predicted insomnia severity and PSA ($\beta = .46$, $p < .001$) significantly predicted insomnia severity. In addition, there was statistically significant indirect effect of PSA as the mediator for IVI predicting insomnia severity (IE = .18, $p < .001$), but not for IVT predicting insomnia severity (IE = .07, $p = .08$).

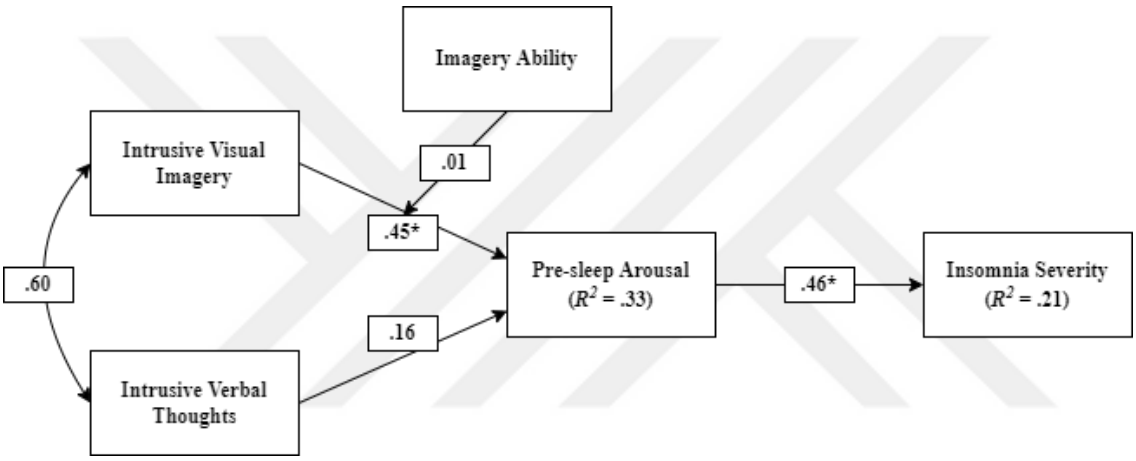


Note 1. * $p < .001$
Note 2. Standardized Path coefficients among variables are presented. .10 = small effect, .30 = medium effect, and .50 = large effect (Cohen, 1992).

Figure 10 The Path Model

3.2.3 Moderation Analysis

To test the hypothesis 5 that VIA has a moderator role on the relationship between IVI and PSA, a moderation analysis was conducted. The model included IVI, VIA, the interaction of IVI and VIA. Moderational effect of VIA on the relationship between IVI and PSA was not significant, $R^2 = .01$, $F\ (1, 162) = 2.56$, $p = .11$. A non-significant moderation effect means that there were no statistical significance transition points within the observed range of the VIA. The hypothesis 5 was not supported. The path model with moderation can be seen on Figure 11.



Note 1. * $p < .001$
Note 2. Standardized Path coefficients among variables are presented. .10 = small effect, .30 = medium effect, and .50 = large effect (Cohen, 1992)

Figure 11 The Path Model with Moderation

CHAPTER 4

DISCUSSION

4.1 Overview of Study 1

The aim of Study 1 was to adapt PSAS and IVIVTQ to Turkish. The underlying factor structures of the scales were examined with EFA and CFA. Convergent and divergent validities of the scales were explored via correlation coefficients between the scales and other related scales. Incremental and Discriminant validities of PSAS were tested with regression analysis and independent t-tests, respectively. Finally, reliabilities of the scales were investigated with internal consistency values and test-retest correlations. In this chapter, the findings of the study were discussed according to existing literature. Furthermore, limitations of the study and future study suggestions were presented.

4.2 Psychometric Properties of Pre-sleep Arousal Scale

In the original scale development study, the factor structure of PSAS was determined by three clinical psychologists who categorized 16 items of PSAS into two factors (Nicassio et al., 1985). Therefore, in the current study, EFA was carried out to determine the factor structure of Pre-sleep Arousal Scale, and CFA was carried out to assess the two-factor structure of the scale.

The results of EFA yielded a two-factor structure that was aligned with the original study, a large community sample study by Janssons-Fröjmark and Norell-Clarke (2012), German (Gieselmann et al., 2012), Pakistani (Namra & Tazvin, 2014), Portuguese (Ruivo Marques et al., 2018) and Japanese (Okajima et al., 2020) adaption studies. All items except Item-8 had higher factor loadings than .40. Item-8 (Ortamdaki ses ve gürültülerden rahatsız olma (örn., saatin tik tak sesleri, evdeki gürültüler, trafik)) did not have sufficient factor loading to be placed in PSAS-C. This

item also did not have sufficient factor loading (.31) in Janssons-Fröjmark and Norell-Clarke's study (2012), did not loaded to PSAS-C in German adaptation study (Gieselmann et al., 2012) and had the lowest factor loading (.37) to PSAS-C in Japanese adaptation study (Okajima et al., 2020). On the other hand, the item had adequate factor loading in Pakistani (.56) (Namra & Tazvin, 2014) and Portuguese (.52) adaptation studies (Ruivo Marques et al., 2018). Considering the rest of PSAS items, all of which refer to negative pre-sleep mental activities, Item-8 having no thought content but an experience of being distracted may be distinguished from the rest of the items. Furthermore, the differences between the studies may be due to the different minimum factor loading thresholds (.30, .32, .35, and 50) utilized in the studies.

The two-factor structure of PSAS with 15 items found in EFA was also assessed by CFA. The results of CFA indicated that the model had an adequate fit to data after adding correlations between the errors of several items. According to Brown (2015, p. 40), correlated errors may be due to "…common assessment methods (e.g., observer ratings, questionnaires); reversed or similarly worded test items; or differential susceptibility to other influences, such as response set, demand characteristics, acquiescence, reading difficulty, or social desirability.". Bollen and Lennox (1991) asserted that correlated errors might even result from items appearing near each other. Examinations of item dyads having correlated errors suggested that correlated errors may be due to the wording of items and appearing near each other for Items 6 and 7 (Düşüncelerinizi durduramama.- Düşüncelerin kafanızda dönüp durması.), while only due to appearing near each other for Items 3-4 and 9-11.

The convergent validity of PSAS was tested by examining correlations between PSAS-C, PSAS-S, RTSQ, BAI-Somatic, and ISI. Although the items of RTSQ are not specific to the pre-sleep period, they pertain to ruminative thinking style and intrusive thoughts and correspond to cognitive mental activity items of PSAS-C. Similarly, BAI-Somatic consists of items that can be regarded as somatic manifestations of anxiety. In this sense, the items of PSAS-S overlap with the items

of BAI-Somatic. As is known, the association between insomnia and PSA has long been argued and studied, so, the correlation between ISI and PSAS was also investigated for convergent validity (Bonnet & Arand, 2010; Nicassio et al., 1985; Tousignant et al., 2019; Yeh et al., 2015). As a result, it can be argued that the high correlations between RTSQ and PSAS-C (r = .72), BAI-Somatic and PSAS-S (r = .67), and ISI and PSAS (.65) supported the convergent validity of PSAS. In terms of divergent validity, it was excepted that GRAS would not be significantly or highly correlated with PSAS-C and PSAS-S (Hubley, 2014). The results indicated, however, that there were small positive correlations between GRAS and PSAS (r = .25), PSAS-C (r = .28), and PSAS-C (r = 15). Rönkkö and Cho (2022) argued that the divergent validity must be assessed by considering context and relevant theories along with statistical tests. For example, although there is a high correlation (r = .99) between biological sex and gender, these are different constructs (Rönkkö & Cho, 2022). In the current study, PSAS measures PSA, while GRAS is a measure of gender role attitudes. Both the context and theories of these constructs are clearly distinct. Therefore, considering the context and theories of the two scales, the small correlations between GRAS and PSAS-C and PSAS-S may support the divergent validity of PSAS.

The incremental validity of PSAS was examined with a regression analysis. The results yielded that PSAS-C and PSAS-S could explain extra variance in insomnia severity beyond depression, anxiety, and stress. Previous studies yielded that insomnia severity, and cognitive and somatic PSAs were significantly associated with depression, anxiety, and stress (Gupta et al., 2018; Jansson-Fröjmark & Norell-Clarke, 2012; Puzino, Amatrudo, et al., 2019; Puzino, Frye, et al., 2019). On the other hand, in the current study, depression and anxiety were not significant predictors of insomnia severity after cognitive and somatic PSAs had been added to the regression model, meaning that cognitive and somatic PSAs may mediate the relationship between depression and insomnia severity or anxiety and insomnia severity. Furthermore, it was found that insomniacs (ISI scores ≥ 8) had significantly higher PSAS-C and PSAS-S scores than non-insomniacs, supporting PSAS's discriminant

validity. The reliability assessment of PSAS was done by internal consistency and test-retest analyses. Cronbach's alpha values of PSAS and its subscales (PSAS, α = .90; PSAS-C, α = .90; PSAS-S, α = 83) were above recommended .70-.80 values (Hoekstra et al., 2019). Similarly, test-retest correlations of a time interval of three weeks (PSAS, r = .90; PSAS-C, r = .90; PSAS-S, r = 83) were above recommended .75 value (Matheson, 2019). In conclusion, the evidence indicated that the Turkish form of PSAS can be regarded as a valid and reliable measure of PSA.

4.3 Psychometric Properties of Intrusive Visual Imagery and Verbal Thoughts Questionnaire

In the original scale development study. The factor structure of IVIVTQ was assessed only by EFA, which yielded a two-factor structure (McCarthy-Jones et al., 2012). In the current study, the psychometric properties of IVIVTQ were examined with both CFA and EFA. The results of EFA initially yielded a three-factor structure. Later, a two-factor structure was tested because the scree plot and the theory regarding two types of intrusive thoughts suggested a two-factor structure. The two-factor structure consisted of items having factor loadings higher than .40. All items loaded to their related factors. There was a high correlation (r = .69) between the two factors. However, the correlation between the factors was below .80 or .85, which are the thresholds for poor discriminant validity (Brown, 2015). Therefore, the results showed that Turkish form of IVIVTQ had a two-factor structure in EFA similar to the original scale and its subscales can adequately measure two related but distinct constructs, namely intrusive visual and verbal thoughts.

Factor structure of Turkish IVIVTQ found in EFA was further assessed by CFA. The results of CFA indicated that the model had an adequate fit to data after adding correlations between the errors of several items. When the items with correlated errors were examined, it may be asserted that correlated errors for Items 1-2, 2-3, 12-13, 15-16 were caused by appearing near each other, while for Items 6-10 (Aklıma gelip duran geçmişime ait olumsuz görüntüler var. - Geçmişime ait olayları istemsizce aklımda görmeye devam ederim.) and Items 16-20 (Aklıma gelip duran

ve geçmişime ait olumsuz sözel düşünceler var. - Geçmişimle ilgili kelime ve ifadeler istemsizce zihnimde yankılanmaya devam eder.) it could be due to wording of items (Bollen & Lennox, 1991; Brown, 2015).

The convergent validity of IVIVTQ was assessed by correlations between IVIVTQ, OSIVQ, DASS, and GRAS. As expected, OSIVQ-Object, measuring VIA, had a stronger association with IVIVTQ-Visual (r = .37) than IVIVTQ-Verbal (r = .17). Furthermore, there were no correlations between OSIVQ-Verbal and IVIVTQ-Visual. Both IVIVTQ-Visual and IVIVTQ-Verbal had high correlations with DASS-D, DASS-A, and DASS-S. The results indicated that the convergent validity of IVIVTQ was supported. In terms of divergent validity, it was excepted GRAS would not be significantly or highly correlated with IVIVTQ-Visual and IVIVTQ-Verbal (Hubley, 2014). The results showed that there were small positive correlations between GRAS and IVIVTQ (r = .23), IVIVTQ-Visual (r = .21), and IVIVTQ-Verbal (r = .22). Acknowledging the context of the scales and the theories of the constructs (intrusive thoughts vs. gender role attitudes) measured by these scales, it could be asserted that the small correlations between GRAS, IVIVTQ-Visual, and IVIVTQ-Verbal supported the divergent validity of PSAS (Rönkkö & Cho, 2022). Due to the limited number of scales in Study 1, incremental and discriminant validities of IVIVTQ were not examined. The evidence showed that the Turkish form of IVIVTQ can be regarded as a valid and reliable measure of IVI and IVT.

4.4 Limitations of Study 1 and Suggestions for Further Studies

The first study was not without its limitations. First of all, the male-female ratio of the samples was biased towards females. Previous studies found that females are more likely to be diagnosed with insomnia disorder (Perlis et al., 2016). For PSAS, it may be possible that the findings reflect the tendencies of females and may have low generalizability. Future studies may use more balanced samples in terms of gender, and investigate factor invariance or other psychometric differences across gender groups. Second, the majority of the participants were university students, which also impairs the generalizability of the findings. Larger and more representative samples

might be used in future studies. Third, the modification indices were used to specify which residual variances in the same latent constructs that are correlated. This leads to neglecting variables that cause the error correlations in the model but are not included in the model, meaning that error correlations between the items are not explained by variables and their relationships in the model (Hermida, 2015).

4.5 The implications of Study 1

In Study 1, PSAS and IVIVTQ were adapted to Turkish. Neither PSAS nor IVIVTQ was previously adapted to Turkish. Previous studies showed that PSAS was a valid and reliable measure to assess PSA. The construct of PSA has significant associations with sleep problems and insomnia (Broman & Hetta, 1994b; Lemyre et al., 2020; Marques et al., 2015; Palagini et al., 2016, 2017; Puzino, Amatrudo, et al., 2019; Yeh et al., 2015). Turkish PSAS can be utilized in experimental and observational sleep medicine studies that examine PSA in Turkish samples. Furthermore, PSAS scores can be used to predict chronic insomnia disorder diagnosis and discriminate chronic insomnia patients from other patients in the Turkish population, as done in the study of Puzino et al. (2019) for the USA population. Finally, PSAS scores may serve as outcome scores for interventions targeting insomnia or sleep problems.

Current measurement tools of intrusive thoughts in the literature consider intrusive thoughts as a single construct. IVIVTQ is the only measurement tool in the literature that distinguishes between IVI and IVT. As previous studies indicated, the effects of visual mental imagery and verbal processing are not equal in terms of their emotional and physiological impacts (Holmes et al., 2008; Holmes & Mathews, 2005; Ji et al., 2016; Lang, 1979; McNeil et al., 1993; Rauch et al., 2004; Vrana et al., 1986). Therefore, Turkish IVIVTQ can be used to evaluate the differential effects of visual and verbal processes in Turkish samples. IVIVTQ can also be used in studies investigating the role of intrusive thoughts in psychopathologies. Moreover, outcomes of interventions targeting intrusive visual mental imagery and verbal thoughts may be measured with IVIVTQ.

4.6 Overview of Study 2

Study 2 tested six hypotheses regarding intrusive thoughts, PSA, and insomnia severity. The first and second hypotheses entailed that IVI and IVT would significantly predict PSA. The third hypothesis was about the differences between the effect sizes of path coefficients from IVI and IVT to PSA. It was predicted that the effect size of IVI would be higher than the effect size of IVT. The fourth hypothesis argued that PSA would significantly predict insomnia severity. The fifth hypothesis suggested that VIA would moderate the relationship between IVI and PSA. The results supported hypotheses 1, 3, and 4. In this section, The findings of Study 2 were discussed according to the current literature. Furthermore, limitations, and clinical implications of Study 2 were presented.

4.7 The Findings Regarding Hypotheses 1, 2, and 3

Although there were studies investigating the relationship between intrusive thoughts and PSA (Tousignant et al., 2019; Yeh et al., 2015), no previous studies examined the difference between predictive powers of IVI and IVT on PSA and insomnia severity in a clinical sample. The findings of the path model showed that IVI, but not IVT, significantly predicted PSA. Intrusive thoughts explained 32.70% of the variance in PSA. The results suggested that hypothesis 1 was supported, while hypothesis 2 was not. The indirect effect of IVI on insomnia severity via PSA was also significant. Moreover, the effect size of path coefficient from IVI to PSA ($\beta = .45$, $p < .001$) was higher than the effect size of path coefficient from IVT ($\beta = .16$, $p = .07$) to PSA, meaning that hypothesis 3 was also supported. These findings supported previous studies (Cuthbert et al., 2003; Holmes et al., 2008; Holmes & Mathews, 2005; Lang, 1979; Vrana et al., 1986), which indicated that visual imagery has a more powerful effect on emotions and arousal levels than verbal processing.

The non-significant path coefficient from IVT to PSA was surprising because it was expected that IVT would significantly predict PSA with a smaller effect size than IVI. Previous studies showed that visual imagery activates frontal cortex areas, hippocampus, primary visual cortex, superior parietal lobule, the supplementary and

cingulate eye fields, and the frontal eye fields (Dijkstra et al., 2017; Pearson, 2019b; Winlove et al., 2018), while verbal thoughts were associated with the left inferior frontal gyrus (Broca's area) and the cingulate cortex activity (Hurlburt et al., 2016; Kühn et al., 2013). Activating brain areas related to visual perception, visual imagery may induce a more realistic simulation of anxiety-provoking events, which evokes stronger psychophysiological responses compared to verbal processing (Ji et al., 2016). Moreover, a non-significant path coefficient may be a result of using a scale assessing trait IVT rather than a scale of pre-sleep IVT.

Two previous studies by Nelson and Harvey (2003a, 2003b) showed that insomniacs had more negative visual imagery in the pre-sleep period compared to good sleepers. Together with the results of the current study, these findings suggest that IVI may exacerbate insomnia severity of insomniacs by eliciting PSA. However, it should be noted that the amount of explained variance (32.70%) in the path model suggested that there may be other important predictors of PSA except intrusive thoughts, such as genetic vulnerabilities, trait hyperarousal, frequency and intensity of arousing events, personal stimulus-arousal associations, poor sleep hygiene, and affect regulation strategies (Espie, 2002; Lundh & Broman, 2000; Palagini et al., 2016, 2017; Spielman et al., 1987).

4.8 The Findings Regarding Hypothesis 4

The path analysis also indicated that PSA significantly predicted insomnia severity, supporting Hypothesis 4. The effect size (β = .45) was medium-large, and PSA explained 21.20% of the variance in insomnia severity. This finding was in line with previous theories (Buysse et al., 2011; Harvey, 2002; Lundh & Broman, 2000; Morin, 1993; Perogamvros et al., 2020; Spielman et al., 1987) that assign a special role to PSA in the formation of insomnia symptoms and the studies showing that higher PSA was associated with higher insomnia severity (Broman & Hetta, 1994b; Marques et al., 2016; Puzino, Amatrudo, et al., 2019; Ruivo Marques et al., 2018; Tousignant et al., 2019; Yeh et al., 2015). Initiation of sleep requires execution of several processes involving cognitive and somatic dearousal, such as inhibition of cortical activity, a

decline in heart rate, a decrease of core body temperature, and increase of distal and proximal skin temperature (Lack et al., 2008; Perlis et al., 1997; Wuyts et al., 2012). Cognitive and somatic PSAs impede normal and involuntary transition from wakefulness to sleep by either eliciting arousal or suppressing dearousal processes (Espie, 2002). The amount of variance in insomnia severity explained by PSA (21.20%) indicated that there might be other important predictors of insomnia severity other than PSA, such as stimulus dyscontrol, poor sleep hygiene, sleep-related worrying, perfectionism, dysfunctional beliefs and attitudes about sleep, and instrumental conditioning (Bootzin, 1972; Espie, 2002; Harvey, 2002; Lundh & Broman, 2000; Perlis et al., 2016; Spielman et al., 1987).

4.9 The Findings Regarding Hypothesis 5

The results showed that there was no moderator role of VIA on the relationship between IVI and PSA. Therefore, Hypothesis 5 was not supported. There could be several reasons for this finding. A previous fMRI study found that there was a strong correlation (r = -.73) between the activation of the visual cortex and the self-report vividness of imagery (Cui et al., 2007). The construct of VIA used in Study 2 does not directly correspond to the vividness of imagery but comprises it (Richardson, 1994). Six items in OSIVQ refer to the vividness of imagery ("When I imagine the face of a friend, I have a perfectly clear and bright image", "My images are very colorful and bright", "When reading fiction, I usually form a clear and detailed mental picture of a scene or room that has been described.", "Sometimes my images are so vivid and persistent that it is difficult to ignore them.", and "My images are very vivid and photographic."). Therefore, it was hypothesized that VIA, including vividness of imagery, would moderate the relationship between IVI and PSA. Yet, it is possible that the interaction effect may not have been represented by the total score of OSIVQ measuring VIA.

Moreover, there may be a ceiling effect in terms of VIA. The participants' lowest score of VIA in the current study was 19, while the lowest score that can be obtained from the VIA subscale is 15. It is possible that the interaction effect may be significant

for extremely low VIA scores between 15-18, which were not obtained in the current study. Finally, a recent imagery extinction study (Hoppe et al., 2022) in which the participants were asked to generate mental imagery of the conditioned stimulus previously paired with electric shock found that high vividness of imagery provided stronger fear reduction during imagery extinction trials compared to the low vividness of imagery. But the long-term results (24 hours later) were not significantly different between high and low vividness imagery groups. The authors (Hoppe et al., 2022, p. 7) commented that: "while high imagery vividness may be advantageous to reduce fear within-session, lower vividness levels may produce comparable long-term effects on fear responses. Patients may only need to surpass a certain threshold of vividness for imaginal exposure to be effective.". According to the results, it could be argued that people with low vividness of imagery can be affected by both positive (fear extinction) and negative (fear learning) outcomes of visual imagery as much as people with the high vividness of imagery, meaning there may be no moderator role for the vividness of imagery.

4.10 Limitations of Study 2 and Suggestions for Further Studies

The second study also had several limitations. First, although path analysis has been believed to prove causality, it can only disprove a model having causal relations among its variables (Streiner, 2005). Experimental and longitudinal studies are needed to be carried out to support the current findings and causal relationships between the variables. Second, identifying the participants with insomnia disorder was done via self-report questions and ISI scores. A more accurate method for diagnosing primary insomnia would be clinical interviews conducted by psychiatrists. Third, most of the sample (75%) was female, young undergraduate students, which impairs the generalizability of the findings to other populations. Future studies may use more representative samples regarding gender, age, and education level. Finally, IVIVTQ-Visual and IVIVTQ-Verbal used in the current study measure IVI and IVT as general traits. A better way to investigate the relationships between IVI, IVT, PSA, and insomnia severity would be using scales

that specifically measure pre-sleep IVI and IVT. There are no such scales in use. Therefore, future studies may be carried out to develop pre-sleep IVI and IVT scales.

4.11 The Clinical implications of Study 2

Several clinical implications of the current study can be proposed. The results indicated that the indirect effects of IVI on insomnia severity through PSA were significant, while the indirect effects of IVT were not. Therefore, interventions reducing pre-sleep IVI may help alleviate sleep problems, while interventions reducing the effects of IVT may or may not. Based on previous studies, several ways to manage pre-sleep intrusive thoughts have been proposed. In this section, previously proposed interventions were discussed, and then a new approach based on the current study and FSMI was asserted.

4.11.1 Eliminating Attempts to Control Intrusive Thoughts

In her meticulous review, Harvey (2005) listed three strategies to deal with intrusive thoughts in insomnia. The first strategy was to eliminate attempts to control intrusive thoughts due to the fact that suppression eventually ends up with the return of suppressed contents (Wegner, 1994). In this sense, Mindfulness meditation can be used to prevent attempts to suppress and control intrusive thoughts (Espie & Ellis, 2016). Mindfulness alludes to "a state of conscious that is characterized by an intentional and non-judgmental awareness of present-moment experiences, rather than attempts to alter current experience or to eliminate them from awareness." (Gong et al., 2016, pp. 1–2). A meta-analysis of six mindfulness meditation for insomnia randomized control trials showed that mindfulness meditation significantly improved only total wake time and sleep quality, but not sleep-onset latency, total sleep time, wake after sleep onset, sleep efficiency, ISI score, Pittsburg Sleep Quality Index score, and Dysfunctional Beliefs and Attitudes about Sleep scale score.

4.11.2 Showing an Effort to Approach Intrusive Thoughts

The second strategy was showing an effort to approach intrusive thoughts (Harvey, 2005). One way of approaching intrusive thoughts may be expressively writing about

them (Lepore, 1997). Harvey and Farrel (2003) asked 42 poor sleepers for three nights to either expressively write about their problems before sleep, distract themselves by writing about their hobbies before bedtime, or not write anything in the pre-sleep period. It was found that expressive and distractive writing groups had shorter sleep-onset latency compared to the control group. But there were no differences between expressive and distractive writing groups in terms of sleep-onset latency. In their study with 28 insomniacs, Mooney et al. (2009) also utilized the expressive writing paradigm. The results showed that expressive writing decreased pre-sleep alertness but not sleep-onset latency.

4.11.3 Distracting Attention Away from Intrusive Thoughts

The third strategy was to distract attention away from intrusive thoughts (Harvey, 2005). In CBT-I, imagery interventions were utilized to induce relaxation or distract the person from intrusive thoughts (Espie & Ellis, 2016; Morin & Azrin, 1987). There were several studies that included visual imagery distraction interventions (Harvey & Payne, 2002; Molen et al., 2013; Morin & Azrin, 1987). Morin and Azrin (1987) compared 21 people with sleep maintenance insomnia who either received stimulus control treatment, imagined six natural objects by focusing their attention on the descriptive features of these objects during day and nighttime awakenings, or were wait-list controls. Both stimulus control and imagery distraction groups had a lower frequency of awakenings and awakening durations compared to the control group. It was also found that stimulus control led to a quicker impact on both measures than imagery distraction. Harvey and Payne (2002) asked three groups of insomniacs (N = 41) for one night to either imagine a pleasant and relaxing situation to distract themselves from unwanted pre-sleep cognitive activity, to simply distract themselves from the pre-sleep cognitive activity without any instruction, or to follow their regular night-time routine. Both imagery distraction and general distraction groups had shorter sleep-onset latency compared to the control group. It was not reported whether there was a significant difference between sleep-onset latency of imagery distraction and general distraction groups.

Molen et al. (2013) examined the effects of reading and imagery distraction interventions added to 3 weeks treatment program, which included lectures on sleep hygiene, dysfunctional beliefs and attitudes about sleep, microanalytic and cognitive models of insomnia, and the role of worries in maintaining insomnia. The participants (N = 80) either listened to guided imagery CD to get rid of pre-sleep worries or read a boring, difficult or calming book for 15 minutes before sleep. There were no differences between cognitive and somatic PSA scores of the reading and imagery distraction groups. Compared to baseline, both groups' cognitive PSA scores decreased significantly, while somatic PSA scores decreased at four weeks follow-up.

4.11.4 Taxing Visuospatial Working Memory by playing Tetris

Previous research has shown that visuospatial tasks occupying working memory may interrupt visual mental imagery (Mertens et al., 2020). One such task is playing Tetris, which may use storage and processing resources in visuospatial working memory and prevent or inhibit IVI (Holmes et al., 2009; Lau-Zhu et al., 2017). Several studies utilizing Tetris showed that it could reduce the occurrence of intrusive traumatic memories (Horsch et al., 2017; Iyadurai et al., 2020; Kanstrup, Kontio, et al., 2021; Kanstrup, Singh, et al., 2021; Kessler et al., 2018, 2020) and strength, vividness and intrusiveness of food and drugs craving imagery (Skorka-Brown et al., 2014, 2015). A recent fMRI study found that playing Tetris activates brain areas associated with visuospatial processing (Agren et al., 2021). In their single case study, Iyadurai et al. (2020) examined the effects of playing Tetris on IVI of a patient with bipolar disorder. Playing Tetris after producing unpleasant visual imagery alleviated vividness, distress, and frequency of IVI. Therefore, it could be argued that playing Tetris can also reduce IVI in the pre-sleep period by taxing visuospatial working memory. Future studies may investigate the effects of playing Tetris (on a non-blue light emitting screen) before sleep on pre-sleep cognitive activity and arousal.

4.11.5 Imagery Rescripting

Although previous studies implemented visual imagery as an intervention, they used visual imagery as a distraction or relaxation method (Schmid & Steil, 2019). In their preliminary study with three insomnia disorder patients, Schmid and Steil (2019) investigated the effects of imagery rescripting on insomnia symptoms. Their treatment module included psychoeducation about insomnia and dysfunctional cognitions, cognitive restructuring of dysfunctional thoughts, and imagery rescripting of the idiosyncratic inner imagery of their sleep difficulties. In the imagery exercise, the participants imagined an aversive image representing their sleep problem and modified it gradually to an alternative non-aversive image. For example, one participant's imagery exercise was reported as following (Schmid & Steil, 2019, p. 238):

> She depicted an earthworm, brown and shining, curled up and stiff similar to metal. The place was cold and there was a sharp smell. She could feel her breath stopping and pressure on her chest. She changed the image to a colourful flowering meadow in the warm sunshine, with blue skies and the fragrance of flowers. The earthworm relaxed and sprawled, became pink in colour and very long, enjoyed the sunshine and moved freely.

Moreover, the participants were required to rehearse the imagery exercise daily for three weeks. The results showed that sleep-onset latency significantly decreased (by 23%), but insomnia severity and sleep quality were unaffected.

4.11.6 Simulated Fear Extinction Intervention

A visual imagery intervention informed by FSMI would target classical conditioning contributing to sleep problems. It is assumed that pre-sleep IVI (US) conditions sleep-related stimuli (CS) with fear arousal (CR). Current models of fear extinction assert that extinction occurs when CS is presented repeatedly in the absence of US (Myers & Davis, 2007). It is also argued that extinction does not eliminate the initial association between CS and US but produces a novel association that inhibits the expression of previous CS-US association (Milad & Quirk, 2012). Furthermore, research suggested that extinction memory is associated with the context in which

extinction is learned, meaning that inhibitory association is activated only when the learner is in the extinction context (Vervliet et al., 2013). For example, if a rat associates a light (CS) in a cage (context) with electric shock (US) and gives a freezing response (CR), fear extinction occurs when the light (CS) is repeatedly presented without electric shock (NoUS). After fear extinction is established, the light does not elicit the rat's freezing response anymore. But, if the rat is placed in a different context, such as a maze, presenting the same light in the maze may activate the freezing response again (CR).

A person who has IVI in the pre-sleep period simulates past or future aversive events (US), and this simulation may evoke fear arousal (UR). If this frequently happens in the acute insomnia period, sleep-related stimuli (CS) such as bed, bedroom, and bedtime are associated with fear arousal (CR). Delayed fear extinction and pre-sleep intrusive imagery, which may constantly consolidate fear learning, may prevent successful fear extinction and transform acute insomnia into chronic insomnia. In the context of insomnia, the sleeping environment can be regarded as the context of fear learning. Therefore, bed, bedroom, and bedtime must be present in absence of IVI to establish fear extinction in sleeping environment. This can be done using visual imagery in which CS-noUS associations are simulated (Mertens et al., 2020; Perogamvros et al., 2020). I call it *simulated fear extinction intervention* (SFEI). There may be several steps for such an intervention. First, in the pre-sleep period, participants lying in bed are asked to imagine themselves in their current sleep environment. Second, they are instructed to imagine themselves peacefully falling asleep, maintaining their sleep, and waking up the next morning in their current sleep environment. This basic visual imagery exercise may help the formation of new CS-noUS associations in which sleep-related stimuli are associated with the absence of IVI (see Figure 12).

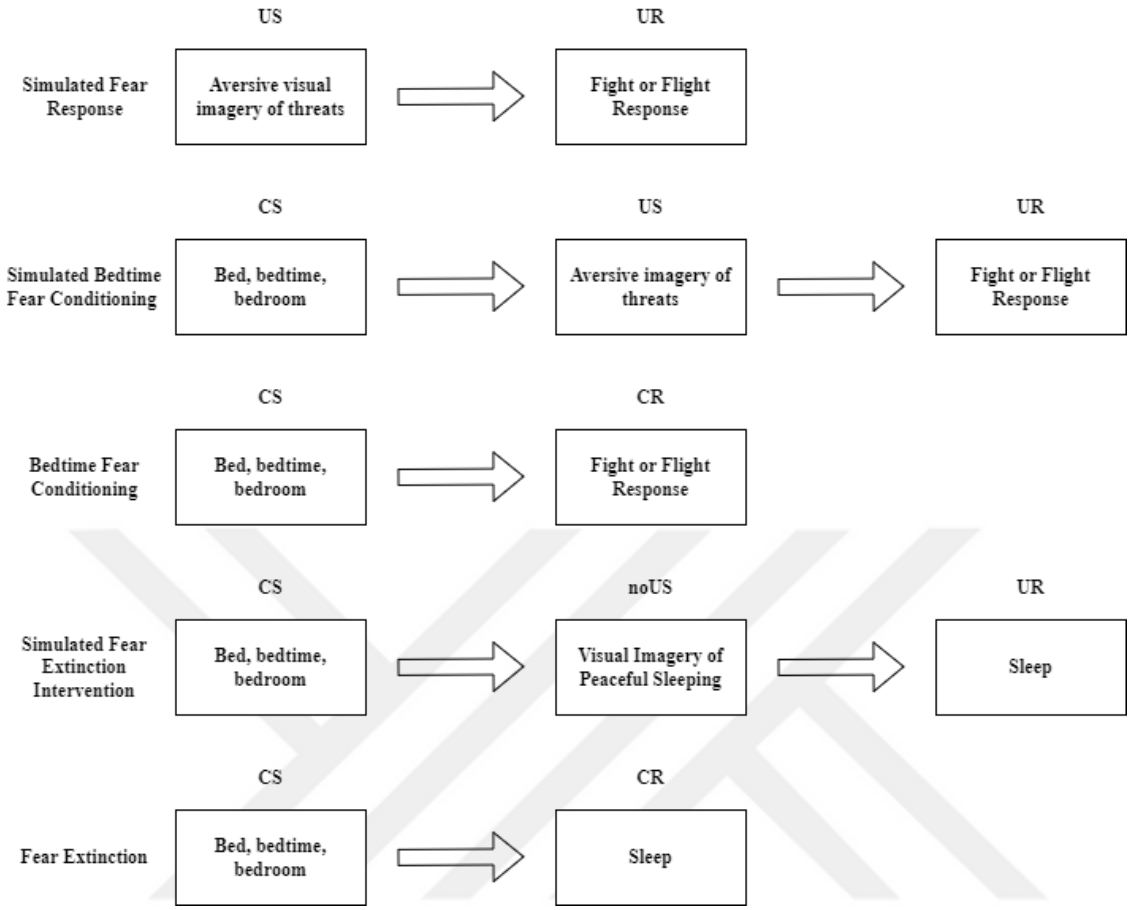


Figure 12 Simulated Fear Extinction Intervention

If the hypothesis that IVI is responsible for bedtime fear conditioning is true, it is plausible that any intervention that prevent IVI may serve as noUS. The proposed SFEI intervention is different from imagery distractions, imagery relaxation, and physical relaxation interventions in many ways. First, all imagery intervention methods, including SFEI, imagery distractions, and imagery relaxation, may inhibit IVI by taxing visuospatial working memory. But only SFEI occurs in the context in which fear conditioning and fear extinction take place and therefore addresses both contextual and cued fear conditioning (Perogamvros et al., 2020). If the person imagines himself or herself in any other environment (e.g., in a peaceful and calming seaside or forest) that is different from his or her bedroom, contextual and cued fear conditioning may not be fully addressed. In this case, the imagined environment

rather than the sleeping environment may serve as the learning context. Due to the fact that the learning context is associated with the inhibitory association, the success of the inhibitory association requires the imagined context to be not different from the sleeping environment. Second, although physical relaxation interventions contribute to inhibition of arousal, they do not deal with IVI. Any IVI at bedtime or during the day may easily reconsolidate fear conditioning. Third, SFEI can be rehearsed during the daytime as it includes the learning context in imagery form. But it should be investigated whether practicing SFEI in the daytime disrupts circadian rhythm by simulating sleeping.

SFEI can be facilitated by the use of visual materials such as photos and videos in which poor sleepers are depicted as sleeping. These materials may help poor sleepers imagine their environment more easily. SFEI can also be applied via virtual reality, which has been shown to be an effective intervention for anxiety-related disorders (Carl et al., 2019). If the person and their sleeping environment can be simulated in virtual reality realistically, virtual reality may substitute visual imagery. The results of the current study suggested that if fear learned by IVI has an important role in the formation of insomnia symptoms and transformation of acute insomnia to chronic insomnia, the proposed intervention will yield much better results than current insomnia interventions. Therefore, further research should be undertaken to compare the differential effects of SFEI, imagery distraction, and imagery relaxation on chronic insomnia.

In previous studies, it has been found that the learned fear can return even after extinction in three ways (Craske et al., 2018; Vervliet et al., 2013). First, it can be reinstated with the presentation of the US without CS. Any IVI that comes to an insomniac's mind, even at a time that is unrelated to sleep, may reinstate learned fear during sleep. Second, contextual renewal can occur as the context in which extinction happened changes. Sleeping in an environment that is different from an insomniac's regular environment may evoke learned fear. Third, a time after extinction without any further learning (SFEI) may bring back fear spontaneously. Vervliet et al. (2013)

and Craske et al. (2018) recommended several ways to overcome such sleep problem relapses, some of which can be applied to prevent relapse in SFEI due to reinstatement, renewal, and spontaneous recovery. First, the number of extinction trials may be augmented dramatically (Vervliet et al., 2013). Augmenting the number of extinction trials in SFEI, however, may increase sleep-onset latency if it is applied in the pre-sleep period. Increasing the number of extinction trials in SFEI may also be achieved by increasing the number of days in which SFEI would be performed. Second, retrieval cues that signal CS-noUS associations can be presented during extinction trials (Craske et al., 2018). For example, a retrieval cue may be an object such as a band or necklace that is worn during SFEI. Third, extinction trials can be conducted in multiple contexts. In terms of SFEI, the person may imagine sleeping not only in his regular sleeping environment but also in other possible sleeping environments.

4.12 Conclusion

In Study 1, PSAS and IVIVTQ were adapted to Turkish. The investigation of psychometric properties of PSAS and IVIVTQ showed that the Turkish forms of the scales were valid and reliable measures to assess pre-sleep arousal and intrusive visual imagery and verbal thoughts. The scales can be used in insomnia and intrusive thoughts research conducted in Turkish speaking populations. In Study 2, a path model including IVI, IVT, PSA, and insomnia severity was tested in a sample of participants who met DSM-V criteria for insomnia disorder. Furthermore, it was examined whether VIA moderates the relationship between IVI and PSA. The results of Study 2 suggested that IVI plays an important role in predicting PSA and insomnia severity, and its effect is not moderated by the levels of VIA. Study 2 pointed out that IVI, which was mentioned only by a few studies in insomnia research, may be an important factor that exacerbates sleep complaints of insomniacs by inducing pre-sleep arousal. Moreover, the lack of moderator role for VIA indicated that both people with high and low VIA might be vulnerable to the negative effects of IVI.

Investigating the role of IVI in insomnia disorder and developing interventions to reduce pre-sleep IVI may bring a better understanding of insomnia disorder and more successful treatments for insomnia disorder. FSMI can be regarded as a nascent insomnia model that requires further empirical testing and development. Similarly, further research is required to establish the therapeutic efficiency of SFEI as a treatment for insomnia disorder, and it must be tested whether it is a better treatment method than existing pharmacological and psychological treatments of insomnia disorder. Then, SFEI can be used as a stand-alone intervention or can be added to CBT-I to address classical conditioning that maintains chronic insomnia. Future studies on the current topic are therefore recommended. Despite the ongoing efforts to explain and treat insomnia disorder, it seems that insomnia disorder will continue to be a serious sleep problem for insomniacs. I hope the present studies contribute to insomnia literature and make us one step closer to more efficient treatments for insomnia disorder.

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APPENDICES

APPENDIX A: APPROVAL OF METU HUMAN SUBJECTS ETHICS COMMITTEE FOR STUDY 1

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29 Eylül 2020

Konu: Değerlendirme Sonucu

Gönderen: ODTÜ İnsan Araştırmaları Etik Kurulu (İAEK)

İlgi: İnsan Araştırmaları Etik Kurulu Başvurusu

Sayın

Danışmanlığını yaptığınız Kutlu Kağan Türkarslan'ın "***Uyku Öncesi Uyarılma Ölçeği ile İstem Dışı Görsel İmgeler ve Sözel Düşünceler Anketi'nin Türkçe Formlarının Psikometrik Özelliklerinin İncellenmesi"*** başlıklı araştırmanız İnsan Araştırmaları Etik Kurulu tarafından uygun görülmüş ve **259-ODTU-2020** protokol numarası ile onaylanmıştır.

Saygılarımızla bilgilerinize sunarız.

APPENDIX B: APPROVAL OF METU HUMAN SUBJECTS ETHICS COMMITTEE FOR STUDY 2

UYGULAMALI ETİK ARAŞTIRMA MERKEZİ
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İlgi : İnsan Araştırmaları Etik Kurulu Başvurusu

Sayın

Danışmanlığını yürüttüğünüz Kutlu Kağan TÜRKARSLAN'ın "Uyku Bozukluğu Belirtileri ile İlişkili Kişilik Özelliklerinin İncellenmesi" başlıklı araştırması İnsan Araştırmaları Etik Kurulu tarafından uygun görülmüş ve **073-ODTU-2021** protokol numarası ile onaylanmıştır.

Saygılarımızla bilgilerinize sunarız.

APPENDIX C: BECK ANXIETY INVENTORY
BECK ANKSİYETE ENVANTERİ

Aşağıda insanların kaygılı ya da endişeli oldukları zamanlarda yaşadıkları bazı belirtiler verilmiştir. Lütfen her maddeyi dikkatle okuyunuz. Daha sonra her maddedeki bugün dahil son bir haftadır sizi ne kadar rahatsız ettiğini yandaki uygun yere (x) işareti koyarak belirleyiniz.

	Hiç 0	Hafif düzeyde *Beni pek etkilemedi* 1	Orta Düzeyde *Hoş değildi ama katlandım* 2	Ciddi düzeyde *Dayanmakta çok zorlandım* 3
1. Bedeninizin herhangi bir yerinde uyuşma veya karıncalanma				
2. Sıcak/ ateş basmaları				
3. Bacaklarda halsizlik, titreme				
4. Gevşeyememe				
5. Çok kötü şeyler olacak korkusu				
6. Baş dönmesi veya sersemlik				
7. Kalp çarpıntısı				
8. Dengenizi kaybedeceğiniz duygusu				
9. Dehşete kapılma				
10. Sinirlilik				
11. Boğuluyormuş gibi olma hissi				
12. Ellerde titreme				
13. Titreklik				
14. Kontrolü kaybetme duygusu				
15. Nefes almada güçlük				
16. Ölüm korkusu				
17. Korkuya kapılma				
18. Midede hazımsızlık veya rahatsızlık hissi				
19. Baygınlık				
20. Yüzün kızarması				
21. Terleme (sıcağa bağlı olmayan)				

APPENDIX D: DEPRESSION ANXIETY STRESS SCALE
DEPRESYON ANKSİYETE STRES ÖLÇEĞİ

Lütfen her bir ifadeyi okuyup 0, 1, 2 veya 3'ten size GEÇEN HAFTA BOYUNCA en uygun olan rakamı yuvarlak içine alınız. Soruların doğru veya yanlış bir cevabı yoktur.

	Hiçbir zaman 0	Bazen 1	Oldukça sık 2	Her zaman 3
1. Sakinleşip rahatlamak bana zor geldi.				
2. Ağzımın kuruduğunu fark ettim.				
3. Hiçbir şekilde olumlu duygular hissedemeyecekmişim gibi geldi.				
4. Nefes alma güçlüğü yaşadım (örn., aşırı derecede hızlı nefes alma, fiziksel egzersiz olmadığı halde nefessiz kalma)				
5. Bir şeyleri yaparken başlamakta zorluk çektiğimi fark ettim.				
6. Olaylara aşırı tepki vermeye meyilliydim.				
7. Titremeler yaşadım (örn., ellerimde)				
8. Bendeki gerginliğin büyük ölçüde enerjimi harcadığını hissettim.				
9. Beni panikletebilen ve kendimi aptal gibi hissedebileceğim durumlardan endişe duydum.				
10. Hiçbir beklentimin olmadığını hissettim.				
11. Tedirgin olduğumu fark ettim.				
12. Rahatlamak bana zor geldi.				
13. Kendimi morali bozuk ve canı sıkkın hissettim.				

14. Yaptığım şeyden beni alıkoyan hiçbir şeye karşı tahammülüm yoktu.				
15. Kendimi paniklemeye yakın hissettim.				
16. Hiçbir şeye karşı bir istek duyamadım.				
17. Bir insan olarak çok fazla değerimin olmadığını hissettim.				
18. Oldukça alıngan olduğumu hissettim.				
19. Fiziksel bir egzersiz yapmadığım halde kalbimin hareketlerini fark edebiliyordum (örn., kalp atış hızında artış hissi, atışlarda düzensizlik)				
20. Ortada bir neden olmadığı halde korktuğumu hissettim.				
21. Hayatın anlamsız olduğunu hissettim.				

APPENDIX E: DEMOGRAPHIC INFORMATION FORM OF STUDY 1
BİRİNCİ ÇALIŞMANIN DEMOGRAFİK BİLGİ FORMU

1) Yaşınız:

2) Cinsiyetiniz:

   a) Kadın
   b) Erkek
   c) Diğer

3) Eğitim seviyeniz **(Öğrenciyseniz şu an eğitim gördüğünüz seviyeyi belirtiniz):**

   a) İlkokul
   b) Ortaokul
   c) Lise
   d) Önlisans
   e) Üniversite
   f) Yüksek Lisans
   g) Doktora

4) Çalışıyor musunuz ?

   a) Hayır
   b) Evet

5) Yaşamınızın büyük bölümünün geçtiği yer:

   a) Büyükşehir
   b) İl Merkezi
   c) İlçe Merkezi
   d) Kasaba
   e) Köy

6) Aylık kazancınızı düşündüğünüzde gelir durumunuzu nasıl tanımlarsınız?

   a) Çok düşük
   b) Düşük
   c) Orta
   d) Yüksek
   e) Çok Yüksek

7) Medeni Durumunuz:

   a) Bekar
   b) Evli
   c) Boşanmış

APPENDIX F: DEMOGRAPHIC INFORMATION FORM OF STUDY 2

İkinci Çalışmanın Demografik Bilgi Formu

1) Yaşınız:

2) Cinsiyetiniz:

   a) Kadın
   b) Erkek
   c) Diğer

3) Öğrenci misiniz?

   a) Evet
   b) Hayır

4) Eğitim seviyeniz **(Öğrenciyseniz şu an eğitim gördüğünüz seviyeyi belirtiniz):**

   a) İlkokul
   b) Ortaokul
   c) Lise
   d) Önlisans
   e) Üniversite
   f) Yüksek Lisans
   g) Doktora

5) Çalışıyor musunuz ?

   a) Hayır
   b) Evet

6) İş çalışma planınız nasıldır?

   a) Sadece gün içinde çalışırım (Örn: 8:30-17:30 arası)
   b) Sadece akşamları çalışırım
   c) 8 saat vardiyalı çalışırım
   d) 12 saat vardiyalı çalışırım
   e) Part-time çalışırım
   f) Diğer

7) Yaşamınızın büyük bölümünün geçtiği yer:

   a) Büyükşehir
   b) İl Merkezi
   c) İlçe Merkezi
   d) Kasaba
   e) Köy

8) Aylık kazancınızı düşündüğünüzde gelir durumunuzu nasıl tanımlarsınız?

a) Çok düşük
b) Düşük
c) Orta
d) Yüksek
e) Çok Yüksek

9) Medeni Durumunuz:

a) Bekar
b) Evli
c) Boşanmış

10) Hiç düzenli olarak sigara kullandınız mı? **(Düzenli = 1 yıl boyunca günde en az 1 sigara)**

a) Hayır
b) Evet

11) Şu an sigara kullanıyor musunuz?

a) Hayır
b) Evet

12) Gün içinde ekran (televizyon, bilgisayar, tablet veya cep telefonu) başında bir şeyler izleyerek ortalama kaç saat geçirirsiniz?

13) Akşamları saat 18:00'den sonra kaç bardak siyah çay içersiniz?

14) Akşamları saat 18:00'den sonra kaç bardak kahve (kafeinli) içersiniz?)

15) Haftada kaç gün egzersiz/spor yaparsınız?

16) Yaptığınız egzersiz/spor aktivitesi ortalama kaç dakika sürer?

17) Yaklaşık olarak ne zamandan beri egzersiz/spor yapıyorsunuz?

Ay:
Yıl:

18)Uyumak için yatağa yattıktan sonra cep telefonuza, tabletinize ya da bilgisayarınıza bakarak ortalama kaç dakika geçirirsiniz?

APPENDIX G: GENDER ROLE ATTITUDES SCALE

Toplumsal Cinsiyet Rolleri Ölçeği

Değerli katılımcı, aşağıda sizinle ilgili ifadeler bulunmaktadır. Lütfen her bir maddeyi dikkatlice okuyunuz ve sizi en iyi tanımlayan seçeneği işaretleyiniz. Doğru ya da yanlış cevap yoktur. Sizden beklenen içtenlikle cevap vererek bilimsel bir çalışmaya yardımcı olmanızdır. Lütfen bütün sorularla ilgili görüşlerinizi ifade ediniz.

	Hiç katılmıyorum 1	Katılmıyorum 2	Kararsızım 3	Katılıyorum 4	Tamamen Katılıyorum 5
1. Erkeklerin ev işlerine yardım etmede kızlarla aynı sorumlulukları vardır.					
2. Ev işleri cinsiyete göre paylaştırılmamalıdır.					
3. Erkek aileden sorumlu olduğu için kadın ona itaat etmelidir.					
4. Bir kadın eşine ters davranmamalıdır.					
5. Bir kadından ziyade bir erkeği ağlarken görmek daha kötüdür.					
6. Kızlar erkeklerden daha derli toplu ve temiz olmalıdır.					
7. Erkekler sadece sorumlu olduğu konularla ilgilenmelidir.					
8. Erkekler kızlara göre farklı şekilde yetiştirilmelidir.					
9. Arkadaş çevreme göre, gelecekteki ev içi faaliyetlerim mesleki faaliyetlerimden daha önemlidir.					

10. Bir babanın temel sorumluluğu maddi konularda çocuklarına yardım etmektir.					
11. Bazı işler kadınlar için uygun değildir.					
12. Arkadaş çevremde eşimin iş geleceği benim iş geleceğimden daha önemlidir.					
13. Çocukların nasıl yetiştirileceğine çoğunlukla annesi karar vermelidir.					
14. Sadece bazı işler kadın ve erkeğe eşit derecede uygundur.					
15. Birçok önemli işte erkeklerle anlaşmak kadınlarla anlaşmaktan daha kolaydır.					

APPENDIX H: INFORMED CONSENT FORM

Bilgilendirilmiş Onam Formu

Bu araştırma, ODTÜ Psikoloji Bölümü öğretim üyelerinden Doç. Dr. Deniz Canel Çınarbaş danışmanlığında, doktora öğrencisi Kutlu Kağan Türkarslan tarafından yürütülmektedir. Bu form sizi araştırma koşulları hakkında bilgilendirmek için hazırlanmıştır.

Çalışmanın Amacı Nedir?

Araştırmanın amacı, uykusuzluk ile bazı kişilik özellikleri arasındaki ilişkilerin incelenmesidir.

Bize Nasıl Yardımcı Olmanızı İsteyeceğiz?

Araştırmaya katılmayı kabul ederseniz, sizden beklenen ankette yer alan bir dizi soruya belirtilen derecelendirme ölçekleri üzerinden yanıt vermenizdir. Bu çalışmaya katılım ortalama olarak 20 dakika sürmektedir.

Sizden Topladığımız Bilgileri Nasıl Kullanacağız?

Araştırmaya katılımınız tamamen gönüllülük temelinde olmalıdır. Ankette, sizden kimlik veya kurum belirleyici hiçbir bilgi istenmemektedir. Cevaplarınız tamamıyla gizli tutulacak, sadece araştırmacılar tarafından değerlendirilecektir. Katılımcılardan elde edilecek bilgiler toplu halde değerlendirilecek ve bilimsel yayımlarda kullanılacaktır. Sağladığınız veriler gönüllü katılım formlarında toplanan kimlik bilgileri ile eşleştirilmeyecektir.

Katılımınızla ilgili bilmeniz gerekenler:

Çalışma, genel olarak kişisel rahatsızlık verecek sorular içermemektedir. Ancak, katılım sırasında sorulardan ya da herhangi başka bir nedenden ötürü kendinizi rahatsız hissederseniz cevaplama işini yarıda bırakıp çıkmakta serbestsiniz.

Araştırmayla ilgili daha fazla bilgi almak isterseniz:

Bu çalışmaya katıldığınız için şimdiden teşekkür ederiz. Çalışma hakkında daha fazla bilgi almak için Kutlu Kağan Türkarslan (E-posta: kutlu.turkarslan@metu.edu.tr) ile iletişim kurabilirsiniz.

APPENDIX I: INSOMNIA SEVERITY INDEX

Uykusuzluk Şiddeti İndeksi

1) Lütfen şu andaki (örn., son 2 hafta içinde) uykusuzluk probleminizin/problemlerinizin **şiddetini** değerlendiriniz.

	Hiç 0	Hafif 1	Orta 2	Şiddetli 3	Çok şiddetli 4
Uykuya dalmakta güçlük					
Uykuyu sürdürmekte güçlük					
Çok erken uyanma problemi					

2) Son zamanlardaki uyku düzeninizden ne kadar memnunsunuz/memnuniyetsizsiniz?

Çok memnun 0	Memnun 1	Nötr 2	Memnun değil 3	Hiç memnun değil 4

3) Uyku probleminizin gün içindeki işlevselliğinizi (örn., gün içinde tükenmişlik, işte/günlük uğraşlarda çalışma potansiyeli, konsantrasyon, hafıza, duygu durum, vb.) ne ölçüde engellediğini düşünüyorsunuz?

Kesinlikle engelleyici değil 0	Biraz engelleyici 1	Oldukça engelleyici 2	Çok engelleyici 3	Çok fazla engelleyici 4

4) Yaşam kalitenizin bozulması anlamında uyku probleminizin başkaları tarafından ne kadar fark edilebildiğini düşünüyorsunuz?

Kesinlikle fark edilmez 0	Biraz fark edilebilir 1	Oldukça fark edilebilir 2	Çok fark edilebilir 3	Çok fazla fark edilebilir 4

5) Son zamanlardaki uyku probleminiz sizi ne kadar endişelendiriyor/strese sokuyor?

Kesinlikle endişelendirmiyor 0	Biraz endişelendiriyor 1	Oldukça endişelendiriyor 2	Çok endişelendiriyor 3	Çok fazla endişelendiriyor 4

APPENDIX J: INTRUSIVE VISUAL IMAGERY AND VERBAL THOUGHTS QUESTIONNAIRES

İstem Dışı Görsel İmgeleme ve Sözel Düşünceler Anketi

Birazdan göreceğiniz sorular zihninizde oluşan, resimler ya da filmlerdeki sahneler şeklinde beliren görüntüler ile ilgilidir. Bu ‘‘zihninizin gözünden’’ görme deneyimidir. Örneğin, bir tatilden sahneler ya da bir ebeveyninizin ya da eşinizin görüntüsü rastgele bir şekilde aklınıza gelebilir. Lütfen aşağıdaki ifadelerden her birinin size ne kadar uygun olduğunu belirtin.

	Kesinlikle katılmıyorum 1	Katılmıyorum 2	Emin değilim 3	Katılıyorum 4	Kesinlikle katılıyorum 5
1. Aklıma gelen ve silemediğim görüntüler var.					
2. Düşüncelerim sıklıkla belli bir görüntüye yönelir.					
3. Aklımda durduramadığım görüntüler var.					
4. Aklıma aniden gelen görüntüler var.					
5. Aklıma sürekli gelen görüntüler sonucu uyumakta zorlanırım.					
6. Aklıma gelip duran geçmişime ait olumsuz görüntüler var.					
7. Biriyle tartıştığımda, tartışma ile ilgili görüntüleri birkaç gün boyunca ve istemediğim halde aklımda görmeye devam ederim.					
8. Gelecekte olacak şeylerin görüntülerini istemsizce ve sık sık kafamda canlandırırım.					
9. Aklıma giren ve kaçınamadığım bazı görüntüler var.					
10. Geçmişime ait olayları istemsizce aklımda görmeye devam ederim.					

Aşağıdaki sorular kelime şeklindeki düşünceleriniz ile ilgilidir. Örneğin, aptalca bir şey yaptığınızda, sanki sessizce kendinize söyler gibi, ‘Ne düşüncesizim’ diye aklınızdan

geçirebilirsiniz. Lütfen aşağıdaki ifadelerden her birinin size ne kadar uygun olduğunu belirtin.

	Kesinlikle katılmıyorum 1	Katılmıyorum 2	Emin değilim 3	Katılıyorum 4	Kesinlikle katılıyorum 5
1. Aklıma gelen ve silemediğim sözel düşünceler var.					
2. Düşüncelerim sıklıkla belli bir kelimeye ya da ifadeye yönelir.					
3. Aklımda durduramadığım sözel düşünceler var.					
4. Aklıma aniden gelen sözel düşünceler var.					
5. Aklıma sürekli gelen sözel düşünceler sonucu uyumakta zorlanırım.					
6. Aklıma gelip duran ve geçmişime ait olumsuz sözel düşünceler var.					
7. Biriyle tartıştığımda, tartışmayla ilgili sözel düşünceleri, istemediğim halde birkaç gün boyunca aklımdan geçirmeye devam ederim.					
8. Gelecekte olacak şeylerle ilgili sözel düşünceleri sık sık ve istemsizce aklımdan geçiririm.					
9. Aklıma giren ve kaçınamadığım bazı sözcük ve ifadeler var.					
10. Geçmişimle ilgili kelime ve ifadeler istemsizce zihnimde yankılanmaya devam eder.					

APPENDIX K: PRE-SLEEP AROUSAL SCALE

Uyku Öncesi Uyarılma Ölçeği

Lütfen aşağıdaki belirtilerden her birini, kendi yatak odanızda uykuya dalmaya çalıştığınız sırada ne yoğunlukta deneyimlediğinizi belirtiniz.

	Hiç 1	Çok az 2	Orta Derecede 3	Fazla 4	Aşırı derece 5
1. Uykuya dalma ile ilgili endişe.					
2. Gün içinde olanları gözden geçirme ya da üzerine düşünme					
3. Depresif ya da kaygı verici düşünceler					
4. Uyku dışındaki sorunlar hakkında endişelenme.					
5. Zihinsel olarak tetikte ve aktif olma.					
6. Düşüncelerinizi durduramama.					
7. Düşüncelerin kafanızda dönüp durması					
8. Ortamdaki ses ve gürültülerden rahatsız olma (örn., saatin tik tak sesleri, evdeki gürültüler, trafik)					
9. Kalp atışlarının hızlanması, küt küt atması ya da düzensiz kalp atışı.					
10. Bedeninizde huzursuz, gergin bir his					
11. Nefes darlığı ya da zor nefes alma					
12. Kaslarınızda kasılma ya da gerginlik hissi.					
13. Ellerinizde, ayaklarınızda ya da vücudunuzun genelinde soğukluk hissi.					
14. Mide rahatsızlığı (midenizde düğümlenme ya da gerginlik hissi, mide yanması, bulantı, gaz vb.)					
15. Avuç içlerinde ya da vücudunuzun diğer yerlerinde terleme					
16. Ağızda ya da boğazda kuruluk hissi					

APPENDIX L: PRIMARY INSOMNIA QUESTIONS

Birincil Uykusuzluk Belirleme Soruları

1) Lütfen varsa aşağıdaki uyku problemlerinden hangilerini yaşadığınızı belirtiniz? **(Birden fazla problemi seçebilirsiniz)**

   a) Herhangi bir uyku problemim yok
   b) Uykuya dalmada güçlük
   c) Uykuyu sürdürmede zorluk
   d) Normal uyanma saatinizden çok daha erken uyanma
   e) Dinlendirici olmayan uyku

2) Uyku probleminiz hayatınızın sosyal, mesleki, eğitimsel, akademik, davranışsal vb. alanlarında ciddi bir zorluk ve bozulmaya neden oluyor mu?

   a) Evet
   b) Hayır

3) Haftada kaç gece uyku problemi yaşıyorsunuz?

   a) 1
   b) 2
   c) 3
   d) 4
   e) 5
   f) 6
   g) 7

4) Uyku probleminiz ne zamandır devam ediyor? **(Size uyan süreyi gösterecek şekilde ay ve yıl göstergelerini uygun sayıya kaydırınız)**

   Ay:
   Yıl:

5) Uyku probleminiz yeterli uyku uyuma imkanınız olmasına (ortalama uyku süreniz kadar uyumanıza izin verecek vaktinizin olması) rağmen mi gerçekleşiyor?

   a. Evet
   b. Hayır

6) Lütfen size konulmuş bir veya daha fazla uyku bozukluğu tanısı varsa belirtiniz:

a. Herhangi bir uyku bozukluğum yok.
b. Varsa tanısını aldığınız uyku bozukluğunu/bozukluklarını aşağıdaki kutucuğa belirtiniz.

7) Lütfen size konulmuş bir veya daha fazla psikiyatrik hastalık tanısı varsa belirtiniz:

a. Herhangi bir psikiyatrik hastalık tanım yok.
b. Varsa tanısını aldığınız psikiyatrik hastalık/hastalıkları aşağıdaki kutucuğa belirtiniz:

8) Lütfen size konulmuş bir veya daha fazla tıbbi hastalık tanısı varsa belirtiniz:

a. Herhangi bir tıbbi hastalık tanım yok.
b. Varsa tanısını aldığınız tıbbi hastalık/hastalıkları aşağıdaki kutucuğa belirtiniz:

9) Lütfen size konulmuş bir veya daha fazla nörolojik hastalık tanısı varsa belirtiniz:

a. Herhangi bir nörolojik hastalık tanım yok.
b. Varsa tanısını aldığınız nörolojik hastalık/hastalıkları aşağıdaki kutucuğa belirtiniz:

10) Lütfen size konulmuş bir veya daha fazla madde bağımlılığı ya da kötüye kullanımı tanısı varsa belirtiniz:

a. Herhangi madde bağımlılığı ya da kötüye kullanımı tanım yok.
b. Varsa tanısını aldığınız madde bağımlılığı ya da kötüye kullanımını aşağıdaki kutucuğa belirtiniz:

11) Lütfen düzenli olarak kullandığınız herhangi bir madde varsa belirtiniz:

a. Herhangi bir madde kullanmıyorum.
b. Varsa kullandığınız maddenin ismini aşağıdaki kutucuğa belirtiniz:

12) Lütfen düzenli olarak kullandığınız herhangi bir ilaç varsa belirtiniz:

a. Herhangi bir ilaç kullanımım yok.
b. Varsa kullandığınız ilacın ismini aşağıdaki kutucuğa belirtiniz:

APPENDIX M: RUMINATIVE THOUGHT STYLE QUESTIONNAIRE

Ruminatif Düşünce Biçimi Ölçeği

Aşağıdaki her bir madde için, ifadelerin sizi ne kadar tarif ettiğini cümlelerin başındaki boşluğa yazınız.

	Hiç 1	2	3	4	5	6	Çok iyi 7
1.Zihnimin sürekli bazı şeyleri tekrar tekrar gözden geçirdiğini fark ederim.							
2.Bir sorunum olduğunda bu durum uzun süre zihnimi kemirir.							
3.Gün boyu bazı düşüncelerin tekrar tekrar zihnime üşüştüğünü fark ederim.							
4.Bazışeyleri sürekli düşünmekten kendimi alamam.							
5.Birileriyle karşılaşma öncesinde olabilecek bütün senaryoları ve konuşmaları zihnimde canlandırırım.							
6.Önceden yaşadığım hoşuma giden olayları hayalimde tekrar canlandırmaya yatkınımdır.							
7.Kendimi, gün içerisinde "Keşke yapsaydım" dediğim şeyleri hayal ederken bulurum.							
8.Kötü geçtiğini düşündüğüm bir görüşme sonrasında, "keşke şöyle davransaydım" dediğim farklı senaryolar hayal ederim.							
9.Karmaşık bir problemi çözmeye çalışırken çözüme ulaşmak yerine problemin başına döndüğümü fark ederim.							
10.Yaklaşan önemli bir olay varsa, bu durumu o kadar çok düşünürüm ki sonunda sinirli ve mutsuz bir hale gelirim.							
11.İstenmeyen düşünceleri zihnimden bir türlü atamam.							
12.Bir problem hakkında saatlerce düşünsem de sorunu açıkça anlamak için biraz daha zamana ihtiyaç duyarım.							
13.Hakkında ne kadar düşünürsem düşüneyim, bazı sorunlarla ilgili net bir çözüme ulaşmam benim için çok zordur.							
14.Bazen bir şeyler hakkında saatlerce oturup düşündüğüm olur.							

15.Bir meseleyi çözmeye çalışırken, zihnimde farklı noktalara dağılan uzun bir tartışma yaşar gibi olurum.							
16.Oturup geçmişteki güzel olayları hatırlamak hoşuma gider.							
17.Heyecan verici bir olayı beklerken, bu olay ile alakalı düşünceler, o anda yaptığım işi engeller.							
18.Bazen, bir konuşma sırasında bile, alâkasız düşüncelerin zihnime hücum ettiği olur.							
19.Yakın zamanda önemli bir görüşme yapacaksam, zihnimde sürekli olarak bunu tekrar etme eğilimim vardır.							
20.Önemli bir olay yaklaşıyorsa bununla ilgili düşünmekten kendimi alamam.							

APPENDIX N: THE OBJECT- SPATIAL IMAGERY AND VERBAL QUESTIONNAIRE

Nesnesel Uzamsal İmgeleme ve Sözel Bilişsel Stil Ölçeği

Aşağıda sizinle ilgili ifadeler bulunmaktadır. Lütfen her bir maddeyi dikkatlice okuyunuz ve sizi en iyi tanımlayan seçeneği işaretleyiniz. Doğru ya da yanlış cevap yoktur. Lütfen bütün sorularla ilgili görüşlerinizi ifade ediniz.

	Katılmıyorum 1	2	3	4	Katılıyorum 5
1. Öğrenciyken 3 boyutlu geometride iyiydim.					
2. Yazı yazarken kendimi ifade etmekte güçlük çekiyorum.					
3. Mühendislik ve görsel sanatlar arasında seçim yapmam istense, mühendisliği seçerim.					
4. Sözel yeteneklerim dil sanatlarında daha kolay meslek edinmemi sağlar.					
5. Mimarlık resim yapmaktan daha çok ilgimi çeker.					
6. İmgelerin çok renkli ve parlaktır. (İmge: Duyularla alınan bir uyaran söz konusu olmaksızın bilinçte beliren nesne ve olaylar.)					
7. Ders kitabı okurken şematik şekilleri ve çizimleri, renkli ve resimsel gösterimlere tercih ederim.					
8. Birçok kişiden daha iyi espri yapar ve öykü anlatırım.					
9. Kompozisyon yazmak bana zor gelir ve yazmaktan hoşlanmam.					
10. İmgelerim ayrıntılı resimlerden çok nesnelerin ve olayların şematik gösterimleri gibidir.					
11. Roman okurken genellikle betimlenen oda ya da sahneyi net ve ayrıntılı biçimde zihnimde oluştururum.					
12. Mühendislik ve görsel sanatlar arasında seçim yapmam istense, görsel sanatları seçerim.					
13. Fotografik bir belleğe sahibim.					

14. 3 boyutlu geometrik şekilleri hayal edebilir ve zihnimde döndürebilirim.					
15. Modern sanatlardaki gibi, parlak ve renkli ve sıra dışı şekillerin olduğu resimlerden hoşlanırım.					
16. Sözel becerilerim mükemmeldir.					
17. Soyut bir kavram (ya da bina) hakkında düşündüğümde, belirli somut bir bina yerine, soyut şematik bir bina ya da onun ayrıntılı tasarımını hayal ederim.					
18. Tanıdık bir dükkâna belli bir şey almak için girdiğimde alacağım nesnenin tam yerini, durduğu rafı, nasıl düzenlendiğini ve çevresindeki eşyaları kolaylıkla zihnimde resmedebilirim.					
19. Ayrıntılı şekil ya da resimler değil de sözel talimatlar verildiğinde mobilya parçalarını (Örn. TV masası ya da sandalye) birleştirmek bana daha kolay gelir.					
20. İmgelerim canlı ve fotografiktir.					
21. Bir şeyler anlatırken, resim ya da çizim yapmaktansa, onu sözel anlatımlarla açıklamayı tercih ederim.					
22. Toplamam için iki basamaklı sayılar (43 ve 32 gibi) verildiğinde, sayıları gözümde canlandırmadan toplamayı kolaylıkla yaparım.					
23. Farklı nesnelere ait zihinsel imgelerim büyüklük, şekil ve renk açısından görmüş olduğum nesnelere çok benzer.					
24. Ders kitabı okurken okuduklarımı genellikle gözümde canlandırmaya ya da çizmeye çalışmam.					
25. Normalde kendiliğinden oluşan canlı görüntüler görmem, çoğunlukla matematikte olduğu gibi bazı problemleri çözmeye					

çalışırken zihinsel imgelemime başvururum. (İmgelem: bir nesneyi o nesne karşımızda olmaksızın tasarımlama yetisi)					
26. Bir arkadaşımın yüzünü hayal ettiğimde, son derece net ve canlı bir görüntü elde ederim.					
27. Teknik grafik konularında mükemmel yeteneklere sahibim.					
28. Bir görüntüyü hatırlarken, zihinsel resimlerden çok sözel betimlemeleri kullanırım.					
29. Bir başkasının kolaylıkla hatırlamayacağı görsel ayrıntıyı; örneğin birinin üstünde gördüğüm gömleğin ya da ayakkabının rengi gibi birçok görsel ayrıntıyı kolaylıkla hatırlayabilir, bazı şeyleri otomatik zihnime alabilirim.					
30. Bildiğim bir binanın ayrıntılı planını kolayca çizebilirim.					
31. Okulda geometri dersiyle hiçbir sorunum olmadı.					
32. Bloklardan ya da kağıtlardan yapılandırmayı gerektiren Lego, Tetris, Origami gibi uzamsal oyunlar oynamakta iyiyim.					
33. Bazen imgelerim göz ardı edilemeyecek derecede canlı ve sürekli olmaktadır.					
34. Gözlerimi kapatıp daha önce yaşadığım bir sahneyi zihnimde kolayca resmedebilirim.					
35. Kelimelerin akıcı kullanmakta ortalamadan daha iyiyim.					
36. Bir nesnenin ya da kişinin resminden çok onun sözel tasvirini tercih ederim.					
37. Cümle yapısına daima hakimimdir.					
38. İmgelerim renkli ve resimselden daha çok şematiktir.					
39. Yazı ve konuşmada çeşitlilik olması için düşüncelerimi farklı biçimlerde ifade edebilmekten hoşlanırım.					
40. Her şeyi görsel olarak hatırlarım. İnsanların ne söylediklerini tartışmaktan çok					

akşam yemeğinde ne giydiklerini, nasıl oturduklarını ve nasıl göründüklerini daha ayrıntılı biçimde hatırlayabilirim.					
41. Bazen söylemek istediğimi ifade ederken sorun yaşıyorum.					
42. Üç boyutlu geometrik bir şeklin döndürüldüğünde nasıl görüneceğini hayal etmek bana zor gelir.					
43. Görsel imgelerim her zaman aklımdadır. Şimdi de oradalar.					
44. Grafiksel yeteneklerim mimarlık alanında daha kolay meslek edinmemi sağlar.					
45. Hiç görmediğim bir radyo spikeri ya da DJ'in sesini duyduğumda, genellikle kendimi onun nasıl göründüğünü resmetmeye çalışırken bulurum.					

APPENDIX O: SECONDARY INSOMNIA EXCLUSION LIST

İkincil Uykusuzluk Dışarıda Bırakma Listesi

Sleep-Wake Disorders:

Obstructive Sleep Apnea, Central Sleep Apnea, Restless Legs Syndrome, Periodic Limb Movement Disorder, Circadian Rhythm Sleep Disorders, Parasomnias.

Mental Disorders:

Major Depressive Disorder, Bipolar Disorder, Dysthymia, Generalized Anxiety Disorder, Panic Disorder, Post-Traumatic Stress Disorder, Obsessive Compulsive Disorder, Schizophrenia, Schizoaffective Disorder, Attention Deficit Hyperactivity Disorder.

Medical Disorders:

Coronary Artery Spasm, Congestive Heart Failure, Dyspnea, Arrhythmia, Chronic Obstructive Pulmonary Disease, Emphysema, Asthma, Laryngospasm, Gastroesophageal Reflux Disease, Peptic Ulcer Disease, Gallstone, Colitis, Irritable Bowel Syndrome, Enuresis, Benign Prostate Hyperplasia, Nocturia, Urinary Tract Infection, Hypothyroidism, Hyperthyroidism, Diabetes, Rheumatoid arthritis, Osteoarthritis, Fibromyalgia, Sjögren Syndrome, Kyphosis, Chronic Kidney Disease, Allergy, Rhinitis, Sinusitis, Bruxism , HIV, Cancer, Scoliosis, Familial Mediterranean Fever, Autoimmune Hepatitis, Menopause, Autoimmune Disease, Cervical Disc Herniation, Multiple Sclerosis, Spinal Disc Herniation, Hashimoto's Thyroiditis, Behçet's Syndrome, Lupus, Pseudotumor Cerebri, Urticaria, Hepatitis, Crohn Disease, Hypertension, Keratoconus.

Neurological Disorders:

Stroke, Dementia, Parkinson’s Disease, Epilepsy, Migraine, Traumatic Brain Injury, Peripheral Neuropathy, Chronic Pain, Neuromuscular disorders, Neurodegenerative Diseases, Fatal Familial Insomnia.

Substance Abuse:

Alcohol, Cannabis, Opioids, Cocaine, Amphetamine, Designer Drugs.

Medications:

SSRIs, Antipsychotics, Allergy/Asthma Medications, Thyroiditis Medications, Stimulants, Decongestants, Painkillers, Opioid Pain Medications, Cardiovascular Medications, Pulmonary Medications, Zoretanin, Beta-Blockers, Diabetic Medications.

APPENDIX P: TURKISH SUMMARY / TÜRKÇE ÖZET

GİRİŞ

Bir İrlanda atasözü şöyle der, "İyi bir kahkaha ve uzun bir uyku bir doktorun çantasındaki en iyi tedavilerdir.". Ne yazık ki bazı insanlar bu basit çarelere erişememektedirler. Uykusuzluğun antik zamanlardan beri insanlar için bir problem olduğu bilinmektedir. Antik Yunan ve Mısır metinlerinde uykusuzluğa ve ona yönelik çarelere pek çok kez değinilmiştir (Asaad, 2015; Montiglio, 2015).

Uykusuzluk genel olarak uyuyamama sorunu olarak kavramsallaştırılmış olsa bile aslında uykuyu başlatma, uykuyu devam ettirme, sabah erken uyanma, uyku süresi ve kalitesi ile ilgili şikayetleri içermektedir. Uzamsal çalışmalar, uykusuzluğun ortaya çıkmasından sonra, şikayetlerin bireylerin %70'de bir yıl boyunca, %50'sinde ise 3 yıl boyunca devam ettiğini göstermektedir (Morin ve Benca, 2012). Önceki araştırmacılar uykusuzluğun sebebini ve uykusuzluk hastalığının süreğenliğini açıklamak için pek çok uykusuzluk modeli ortaya koymuşlardır (Perlis ve ark., 2016). Bu çalışmanın amacı birincil uykusuzluğun oluşumunda istenmeyen görsel imgelerin rolünü vurgulayan bir modeli test etmektir.

1.1 Uykusuzluk Bozukluğu'nun Tanımı

Uykusuzluk çekenler temelde üç uyku probleminden mustariptirler: (1) uykuya dalamama, (2) uykuyu sürdürememe ve (3) sabah erken uyanmak (Reeve ve Bailes, 2010). Uykusuzluk problemi çekmeyenler genellikle 20 dakika içinde uykuya dalabilirken (Thomas ve Anderson, 2013), bu süre uykusuzluk çekenler için daha uzundur. Uykusuzluk çekenlerin gece uykuları sık sık bölünür ve onlar uykuya kolayca geri dönemezler. Son olarak, uykusuzluk çekenlerin bir kısmı sabahları normal uyanma saatlerinden çok daha erken uyanmaktadırlar. Tüm bu uyku zorlukları sebebiyle uykusuzluk çekenler düşük uyku kalitesi ve süresi, günlük işlevsellikte bozulmalar, genel stres ve düşük iyilik halinden şikayetçidirler (Morin

ve Benca, 2012; Winkelman, 2015). Uykusuzluk eğer diğer uyku bozuklukları, fiziksel ya da ruhsal rahatsızlıklardan kaynaklanmıyorsa birincil uykusuzluk, eğer kaynaklanıyorsa ikincil uykusuzluk olarak değerlendirilir (Mai ve Buysse, 2008). Ruhsal Bozuklukların Tanısal ve Sayımsal Elkitabı-5 (RBTSE-5) üç alt uykusuzluk tipini içermektedir. Bunlar akut, epizodik ve kalıcı uykusuzluklardır (Amerikan Psikiyatri Derneği, 2013). Uluslararası Uyku Bozuklukları Sınıflandırması-3 (UUBS-3) ise kısa süreli (üç aydan az süredir) ve kronik uykusuzluk (üç aydan fazla süredir) olmak üzere iki tip uykusuzluk içermektedir (Grima ve ark., 2019).

1.2 Uykusuzluk Bozukluğu Tanısı

Uykusuzluk bozukluğu tanısı koymak için en sık kullanılan tanısal araçlar UUBS-3 ve RBTSE-5'dir. RBTSE-5'göre sürekli uykusuzluk tanısı için uykuya dalamama, uykuyu sürdürememe ve sabah erken uyanma gibi uyku problemlerinden en az biri, haftada en az 3 gün görülmeli ve bu problemler en az 3 aydır devam ediyor olmalıdır. Buna ek olarak uyku problemleri ciddi bir strese sebep olup kişinin hayatın farklı alanlarındaki işlevselliği azaltmalı ve uyku problemleri kişinin uyumak için yeterli zamanı olmasına rağmen mevcut olmalıdır. Ayrıca uyku problemlerinin diğer uyku bozuklukları, fiziksel ya da ruhsal rahatsızlıklar veya ilaç ve madde kullanımı tarafından açıklanamıyor olması gerekmektedir. UUBS-3 kronik uykusuzluk tanı kriterleri RBTSE-5 tanı kriterlerine büyük oranda benzemektedir (Amerikan Uyku Tıbbı Akademisi, 2014). Uykusuzluk semptomlarında RBTSE-5'e ek olarak *uygun bir programa göre yatağa gitmeye direnç* gösterme ve *ebeveyn ya da bakım veren müdahalesi olmadan uyumakta zorlanma gibi* ek semptomlar bulunmaktadır. Ek tanı kriterlerinden bir tanesi yorgunluk, dikkat kaybı, hayatın çeşitli hayatlarında performans kaybı ve duygu durumun bozulması, gün içinde uykusuzluk, düşük motivasyon vb. gibi uykusuzluk ile ilişkili sorunlara sahip olmaktır. Diğer tanı kriterleri RBTSE-5 ile aynıdır (Amerikan Uyku Tıbbı Akademisi, 2014).

1.3 Uykusuzluğun Yaygınlığı

Uykusuzluk bozukluğunun yaygınlığı bozukluğun tanımına ve tanı kriterlerinin katılığına göre değişmektedir (Bos ve Macedo, 2019; Ohayon, 2002). Şimdiye kadar

Amerika, Çin, Fransa, Güney Kore, Kanada, İspanya ve Norveç'te yapılan yaygınlık çalışmaları yetişkin popülasyonunun %5-19.2'sinin uykusuzluk bozukluğu için RBTSE-IV tanı kriterlerini sağladığını göstermektedir (Cao ve ark., 2017; Ford ve ark., 2015; Leger ve ark., 2000; Morin ve ark., 2011; Ohayon ve Hong, 2002; Ohayon ve Sagales, 2010; Pallesen ve ark., 2001). Benbir ve arkadaşları (2015) Türkiye'deki uykusuzluk yaygınlığını %12.2 olarak bulmuşlardır.

1.4 Uykusuzluğun Sonuçları

Uykusuzluğun hayat kalitesinin ve günlük işlevselliğin düşmesi, psikomotor ve bilişsel becerilerde bozulma, düşük iş performansı, daha yüksek devamsızlık ve uykusuzluk dışındaki rahatsızlıklar için artan tedavi maliyetleri gibi sonuçları bulunmaktadır (Daley ve ark., 2009; Fortier-Brochu ve ark., 2012; Godet-Cayré ve ark., 2006; LeBlanc ve ark., 2007; Léger ve ark., 2012; Reynolds ve Ebben, 2017; Sofi ve ark., 2014; Stoller, 1994; Wade, 2010).

1.5 Uykusuzluk ve Psikopatoloji

Geçmiş çalışmalar uykusuzluğun psikopatoloji geliştirmede bir risk etmeni olduğunu ortaya koymaktadır. Ohayon ve Roth (2003) uykusuzluğun duygudurum bozukluklarının %41'inde ve kaygı bozukluklarının %18'inde, bu rahatsızlıklardan önce ortaya çıktığını bulmuşlardır. Majör depresif bozukluk hastalarının %80'nin ek uykusuzluk tanısı olduğu ve bu hastaların %50'sinde uykusuzluğun depresyondan önce başladığı görülmektedir (Ohayon ve Roth, 2003). Uykusuzluk majör depresyon geliştirme riskini 2.8 kat, kaybı bozukluğu geliştirme riskini 3.23 kat, alkol kötüye kullanımı riskini 1.35 kat ve psikoz riskini 1.28 kat arttırmaktadır (Hertenstein ve ark., 2019).

1.6 Uykusuzluk Bozukluğu'nun Tedavisi

Uykusuzluk Bozukluğu'nun tedavileri temel olarak farmakoterapi ve psikoterapi olmak üzere ikiyi ayrılmaktadır (Qazi ve Schluederberg, 2012; Reeve ve Bailes, 2010). Uykusuzluk Bozukluğu'nun farmakoterapisinde kullanılan ilaçlar ve maddeler benzodiazepin reseptör agonistleri, melatonin agonisti ramelteon, trisiklik

antidepresan doxepin, barbetüratlar, oreksin reseptör antagonisti suvoreksant, antidepresanlar, atipik antipsikotikler, antihistaminikler ve melatonindir (Lie ve ark., 2015). Buscemi ve arkadaşları (2007) 105 çalışmayı içeren meta-analizde benzodiazepinlerin, benzodiazepin olmayan etken maddelerin ve antidepresanların uykuya geçiş süresini anlamlı olarak azalttığını bulmuşlardır.

Uykusuzluk Bozukluğu'nun tedavisindeki başlıca psikoterapi yöntemi Uykusuzluk için Bilişsel Davranışçı Terapi'dir (BDT-U). BDT-U ortalamada 4 ile 8 seans arasında sürer ve uyku kısıtlaması, uyaran kontrolü, bilişsel yeniden yapılandırma, uyku hijyeni ve rahatlama eğitimi gibi bileşenleri içermektedir (Mitchell ve ark., 2012; Siebern ve ark., 2012). Van der Zweerde ve arkadaşları (2019) 30 seçkisiz kontrollü çalışmayı içeren meta-analizlerinde BDT-U'nun uykusuzluk şiddeti üzerindeki etki büyüklüğünü (Hedge's g) 3.ayda .64, 6. ayda .40 ve 12.ayda .25 olarak bulmuşlardır.

1.7 Uykusuzluk ile İlişkili Sosyodemografik, Fizyolojik ve Psikolojik Değişkenler

1.7.1 Sosyodemografik Değişkenler

Daha önce gerçekleştirilen çalışmalar toplumsal cinsiyetin, yaşın, medeni halin ve sosyo-ekonomik değişkenlerin uykusuzluk bozukluğu ile ilgili olduğunu göstermiştir. Çalışmalar erkeklere kıyasla kadınların daha fazla uykusuzluk belirtisi gösterdiği ve uykusuzluk bozukluğu tanısı aldıklarını (Benbir ve ark., 2015; Ford ve ark., 2015; Käppler ve Hohagen, 2003; Leger ve ark., 2000; Morin ve Benca, 2012; Ohayon, 2002; Ohayon ve Hong, 2002; Pallesen ve ark., 2001; Sivertsen ve ark., 2009); ileri yaşın uykusuzlukla ilişkili olduğunu (Ford ve ark., 2015; Klink ve ark., 1992; Ohayon ve Hong, 2002; Pallesen ve ark., 2001; Sivertsen ve ark., 2009); bekarların evlilere ve boşanmış kişilere kıyasla daha az uykusuzluk raporladıklarını (Benbir ve ark., 2015; Kawata ve ark., 2020; Leger ve ark., 2000); düşük sosyo-ekonomik seviyenin uykusuzluğu yaş, toplumsal cinsiyet ve etnik kimlik kontrol

edildiğinde dahi anlamlı olarak yordayabildiğini bulmuştur (Gellis ve ark., 2005; Sivertsen ve ark., 2009).

1.7.2 Fizyolojik Değişkenler

Fizyolojik değişkenleri içeren çalışmalar uykusuzluk çekenlerin bozuk beden sıcaklık ritimlerine, daha yüksek kortizol seviyesine, daha güçlü elektrodermal aktiviteye ve daha yüksek metabolik hıza sahip olduklarını göstermiştir (Broman ve Hetta, 1994; Chapman ve ark., 2018; Dressle ve ark., 2022; Lack ve ark., 2005; Lack ve ark., 1996; Morris ve ark., 1990).

1.7.3 Psikolojik Değişkenler

Psikoloji açısından incelendiğinde uykusuzluk çekenlerin kaliteli uyku uyuyanlara kıyasla daha yüksek zarardan kaçınma özelliği gösterdikleri görülmektedir (Bravo-Ortiz ve ark., 2013; de Saint Hilaire ve ark., 2005). Lundh ve Broman (2006) kontrol katılımcılarına kıyasla uykusuzluk çekenlerin daha yüksek aleksitimi skorlarına sahip olduklarını bildirmişlerdir. Gurtman ve arkadaşları (2014) uykusuzluk çektiğini belirten katılımcıların iyi uyuyanlara kıyasla daha yüksek nevrotiklik ve deneyime açıklık skorlarına sahipken daha düşük titizlik gösterme skoruna sahip olduğunu belirtmişlerdir. Deker ve arkadaşları (2017) uykusuzluk şiddeti ile nevrotiklik, yumuşak başlılık ve deneyime açık olma kişilik özellikleri arasında olumlu ilişki olduğunu bulmuşlardır.

1.8 Uykusuzluk Modelleri

Uykusuzluk bozukluğunun en ayırıcı özelliklerinden bir tanesi, onu farklı bakış açıları ile açıklamaya çalışan pek çok kuramsal modelin olmasıdır. Bu modellerin bir kısmı koşullanma ya da kognitif değerlendirmeler gibi psikolojik ve davranışsal etmenleri ön plana çıkarırken, diğerleri ise uykusuzluk ile bağlantılı fizyolojik ya da nörolojik değişiklerin önemini vurgulamıştır. Hem psikolojik hem de fizyolojik değişkenlere dayanan modeller ise uykusuzluğun daha bütüncül bir resmini ortaya koymaya çalışmıştır.

1.8.1 Uyaran Kontrolü Modeli (1972)

Bootzin (1972) tarafından geliştirilen Uyaran Kontrolü Modeli, uykusuzluğun oluşmasında edimsel koşullanmanın rolünü açıklamaya çalışmıştır. Eğer bir uyaran sürekli olarak tek bir davranışla eşleştirilirse, bu uyaranın kendisi ile eşleştirilen davranışı ortaya çıkarma olasılığı artmaktadır (Ayırıcı Uyaran – APA Psikoloji Sözlüğü, n.d.; Perlis ve ark., 2016). Bu modele göre uykuya dalmak edimsel bir eylem iken uyku bu davranışın ödülüdür. Bu bağlamda uyku ile ilişkili uyaranlar ödül olan uyku davranışı için ayırıcı uyaran işlevi görmektedir. Yatakta yemek yemek, ders çalışmak ya da TV izlemek gibi yatağı ve uyku ile ilgili uyaranları uyku dışı davranışlar ile ilişkilendirecek eylemler uykuya dalma güçlüklerine sebep olabilmektedir (Bootzin, 1972).

1.8.2 Spielman Modeli (3P Modeli) (1987)

Üç-Etken Modeli olarak da bilinen Spielman modeli uykusuzluğu zemin hazırlayan, tetikleyici ve devam ettiren etmenlerin etkileşimi ile açıklamaya çalışmaktadır (Spielman ve ark., 1987). Zemin hazırlayan etmenler genetik hassasiyetler, aşırı-uyarılabilirite, sürekli endişe ve ruminasyon ve zorunlu bir uyku programına sahip olma gibi değişkenleri içermektedir. Tetikleyici etmenler akut uykusuzluğa sebep olan stresli yaşam olayları, fiziksel ve ruhsal hastalıkları içermektedir (Perlis ve ark., 2016). Son olarak devam ettiren etmenler uyku problemlerine yönelik olarak ortaya ancak uyku problemlerini daha da arttırıp akut uykusuzluğu kronik uykusuzluğa çeviren durum ve davranışları içermektedir. Uyku vaktinin tahmin edilemez olması, uyanık iken yatakta vakit geçirmek, artan kafein tüketimi, yatağa uyku gelmeden gidilmesi, yataktan geç çıkılması ya da gün içinde yapılan kısa kestirmeler bu etmenlere dahil edilebilir (Perlis ve ark., 2016; Spielman ve ark., 1987).

1.8.3 Mikroanalitik Model (1993)

Morin (1993) tarafından geliştirilen Mikroanalitik Model, uykusuzluğun 4 farklı etmenin birbirlerini etkilemesi üzerinden açıklamaya çalışmaktadır. Bu etmenler uyarılma, işlevsel olmayan bilişler, uyum bozucu alışkanlıklar ve sonuçlardır. Uyarılma bilişsel, duygusal ya da fizyolojik olabilir ve yaşamla bağlantılı etmenler

sonucu ortaya çıkmaktadır. İşlevsel olmayan bilişler endişelenmeyi, ruminasyonu, inançları ve uyku problemleri ile ilgili beklentileri içermektedir. Uyum bozucu alışkanlıklar uyku eksikliğinin etkilerini telafi etmek için gerçekleştirilen yatakta fazla vakit geçirme ya da gün içinde kestirme gibi davranışlardır. Sonuçlar ise uykusuzluk sonucu olarak ortaya çıkan gün içinde performans kaybı, yorgunluk ve duygudurum düzenleme problemleri gibi psikolojik ya da sosyal sıkıntıları içermektedir (Morin, 1993).

1.8.4 Nörokognitif Model (1997)

Nörokognitif Model Perlis ve arkadaşları (1997) tarafından 3P modeli ve klasik koşullanmanın uykusuzluğun ortaya çıkmasında ve devam etmesindeki rolünü açıklamak için geliştirilmiştir. Bu modelde uyarılma kortikal, bilişsel ve bedensel olarak kavramsallaştırılmaktadır. Kortikal uyarılma EEG aktivitelerindeki artışa karşılık gelirken bilişsel uyarılma olumsuz tondaki zihinsel aktiviteleri tanımlar. Bedensel uyarılma ise nefes almakta zorlanma, bedensel gerginlik ve kalp atış hızının artması gibi fizyolojik uyarılma belirtileridir. Nörokognitif Model'e göre kortikal uyarılma klasik koşullanma sonucu ortaya çıkan artmış duyusal ve bilgisel işlemleme ve uzun süreli bellek işlevlerinin sonucudur. Uyku ile ilişkili yatak odası, yatak ve uyku zamanı gibi uyaranların tetiklediği kortikal aşırı uyarılma uykunun başlatılmasını engellemektedir (Perlis ve ark., 1997).

1.8.5 Tehdit Algısının Yüksek Risk Modeli (1998)

Perlstrom ve Wickramasekera tarafından geliştirilen Tehdit Algısının Yüksek Risk Modeli uykusuzluğu bedenselleştirme ve tehdit algısı üzerinden açıklamaya çalışmaktadır. Bu modele göre uykusuzluk için risk etmenlerinden bir tanesi hipnotik yetenektir. Yüksek ve düşük hipnotik yeteneği olan insanlar uykusuzluk geliştirmeye daha yatkındırlar (Perlstrom ve Wickramasekera, 1998). Yüksek hipnotik yeteneği olan kişiler tehditlere karşı aşırı duyarlı ve aşırı reaktif oldukları için, düşük hipnotik yeteneği olan kişiler ise duygusal tehditlere az duyarlı ve kronik stres halinde parasempatik düzenleme bozukluğu yaşadıkları için uykusuzluğa meyillidirler. Modele göre diğer bir risk etmeni hipotalamik-hipofiz-adrenal ekseni ve bağışıklık

işlevini, algı ve hafızadaki kronik olumsuz yanlılıklar ile etkileyen yüksek belirgin ve örtük olumsuz duygulanımdır (Perlstrom ve Wickramasekera, 1998).

1.8.6 Uykuyu Bozan-Yorumlayan Süreç Modeli (2000)

Lundh ve Broman (2000) tarafından geliştirilen model, uykusuzluğu uykuyu-bozan ve uykuyu-yorumlayan süreçler üzerinden ele almaktadır. Uykuyu-bozan süreçler fizyolojik, duygusal ve bilişsel uyarılma ve uyarılma seviyesini etkileyen uyarılabilirlik, uyarıcı olayın sıklığı ve yoğunluğu, bireysel duygu düzenleme örüntüleri, kişisel uyaran-uyarılma ilişkileri, davranışlar ve bilişsel başa çıkma stratejileri ve kişiler arası ilişkilerdir. Uykuyu-yorumlayan süreçler ise uyku ve gündüz işlevselliği ile ilgili değerlendirmeleri ve bu etmenleri etkileyen düşük kaliteli uyku ve gündüz işlevselliği ile ilgili değerlendirmeleri, mükemmeliyetçilik seviyesini, uyku ihtiyacı ve düşük kaliteli uykunun sonuçları ile ilgili inançları içermektedir. Uykuyu-bozan süreçler uykunun başlamasını ve sürdürülmesini engellerken, uykuyu-yorumlayan süreçler bireylerin uyku problemlerini uykusuzluk olarak yorumlama ve bu şekilde ifade etmelerine sebep olur (Lundh ve Broman, 2000).

1.8.7 Psikobiyolojik Baskılama Modeli (2002)

Espie (2002) tarafından geliştirilen Psikobiyolojik Baskılama Modeli uyarılmayı azaltan süreçlerin baskılanmasının normal uykuyu nasıl etkilediğini açıklamaktadır. Uykusuzluk genelde bir aşırı uyarılma bozukluğu olarak görülmekteyken bu model aşırı uyarılma üzerindeki vurguyu uyarılmayı azaltan süreçlerin baskılanmasına kaydırmaktadır. Uyarılmayı azaltan süreçler uyku-uyaran kontrolü, fizyolojik uyarılma azalması, bilişsel uyarılma azalması, duygu düzenleme ve gece uykusunun gündüz kolaylaştırılmasını içermektedir. Bu modele göre uyku otomatik (uykunun kendiliğinden başlaması ve devam etmesi) ve plastisiteye (dışsal durumlara adapte olabilmesi) sahip bir şekilde gerçekleşmektedir (Espie, 2002). Akut uykusuzluk uyarılmayı azaltan süreçlerin baskılanması sonucu ortaya çıkarken, kronik uykusuzluk kişinin dikkatini zorlayıcı yaşam olaylarından uykusuzluğa kaydırması ve uykuyu kendi niyeti ile başlatması sonucu açığa çıkar.

1.8.8 Bilişsel Model (2002)

Bilişsel Model, Harvey (2002) tarafından kaygı bozukluklarının bilişsel kuramının bir eklentisi olarak geliştirilmiştir. Ruminasyon ve endişeyi içeren bilişsel uyarılma normal uykuyu engelleyerek kronik uykusuzluğun ortaya çıkma ihtimalini arttırmaktadır. Modele göre uykusuzluk kişi uyku hakkında endişelenmeye başladığında ve uykusuzluğun gün içinde yarattığı eksikliklere yönelik bozulmuş bir algılama geliştirmeye başladığında ortaya çıkar. Kişi uykunun zamanı, uzunluğu, kalitesi ve uykusuzluğun olumsuz sonuçları hakkında endişelendiğinde bilişsel uyarılma ve sıkıntı yaşamaya başlar (Harvey, 2002). Daha sonra uyku ile ilgili tehditlere karşı duyarlı hale geldikçe uyku ile ilgili tehditlere dikkat etmeye ve onları kontrol etmeye başlar.

1.8.9 Nörobiyolojik Model (2011)

Buysse ve arkadaşları (2011) tarafından geliştirilen Nörobiyolojik Model, REM olmayan uykudaki uyanıklığa teşvik eden nöral yapıların aktivesinin uykusuzluk belirtilerini ortaya çıkardığını belirtmektedir. Model hayvan çalışmaları ve uykusuzluk çekenler ile iyi uyuyanlar arasındaki bölgesel nöral aktivite ve metabolizma farklarına dayanmaktadır. Yapılan çalışmalar uykusuzların REM olmayan uykularının prefrontal korteks, paralimbik korteks, thalamus-parietal korteksler ve hipotalamik-beyin sapı bölgelerindeki uyanıklık benzeri nöral aktiviteleri içerdiğini göstermektedir (Buysse ve ark., 2011; Perlis ve ark., 2016). Bu modele göre uykusuzluk hem uyku hem de uyanıklığa yönelik beyin faaliyetlerinin olduğu hibrit bir uyku durumudur.

1.8.10 Mevcut Uykusuzluk Modellerinin Sınırlılıkları

Perlis ve arkadaşları (2016) mevcut uykusuzluk modellerini gözden geçirdikleri değerlendirmelerinde, modellerin yeterli açıklamayı getiremediği altı noktaya değinmişlerdir. Birincisi çoğu uykusuzluk modeli uykusuzlukta klasik koşullanmanın rolünü göz ardı etmektedir. İkincisi çoğu model normal uyku-uyanıklık düzenlemesini kuramsal açıklamalarına dahil etmemektedir. Üçüncüsü bazı modeller akut uykusuzluğun kronik uykusuzluğa tam olarak nasıl dönüştüğünü

açıklamamakta ve elde yeterli kanıt olmamasına rağmen kronik uykusuzluğu akut uykusuzluğun uzamış bir versiyonu olarak değerlendirmektedir. Dördüncüsü, çoğu model kadınların ve ileri yaşlı yetişkinlerin uykusuzluktan neden daha çok mustarip olduklarını açıklamamaktadır. Beşincisi, mevcut uykusuzluk modelleri uyku başlangıcı, gecenin ortası ve sabah erken uyanma gibi uykusuzluk türleri arasında bir ayrım yapmamaktadır. Altıncı olarak çoğu model açık kanıtlar olmamasına rağmen hem akut hem kronik uykusuzluğu aşırı uyarılmaya bağlamakta ve akut ve kronik uykusuzluktaki aşırı uyarılmaların fizyolojik ve psikolojik olarak aynı olduğunu varsaymaktadır.

1.9. Uyku ve Uykusuzluk: Evrimsel Bakış Açısı

Neden uyuduğumuzu açıklamaya çalışan pek çok hipotez olsa da uykunun temel sebebi gizemini korumaktadır (Lee Kavanau, 2005; Nunn ve ark., 2016). Pek çok primat türü ağaçlarda uyurken, ilk olarak büyük maymunlar uyku platformları ve ağaçlar üzerinde barınaklar inşa etmeye başlamıştır (Covert, 1995; Duda ve Zrzavý, 2013). Uyku platformları büyük maymunların daha az uyarılmalarına ve uykularının daha az bölünmesine (Samson ve Shumaker, 2013) ve de artan bir bilişsel kapasiteye sahip olmalarına neden olduğu düşünülmektedir (Nunn ve ark., 2016). Günümüzün endüstri sonrası döneminde yaşayan insanlar, geçmişe ait pek çok tehditten uzak bir hayat sürmelerine rağmen kısalan uyku süreleri ve uyku kalitesi ile ilgili problemler yaşamaktadırlar.

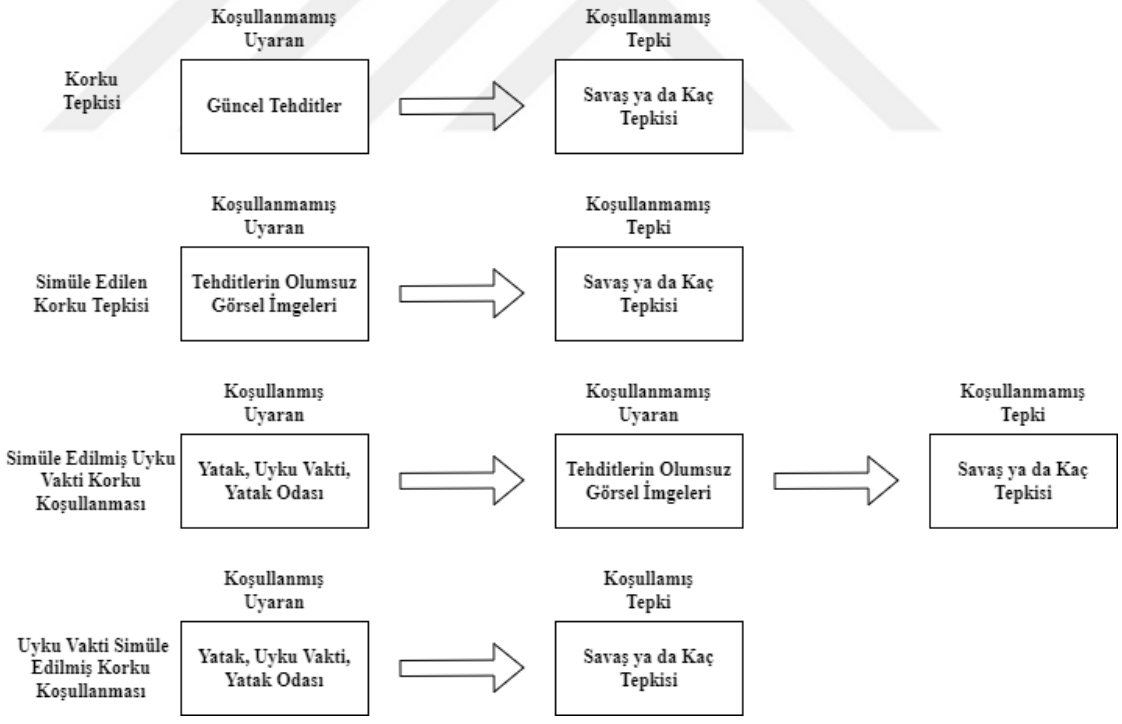
Uykusuzluğu evrimsel bir bakış açısı ile inceleyen McNamara ve Auerbach (2001), uykusuzluk ile ilgili üç temel noktaya işaret etmektedir. İlk olarak uykusuzluk çeken kişiler ile uyku yoksunluğu çeken kişiler karşılaştırıldığında, uykusuzluk çekenlerin ertesi gün öznel olarak uykululuk hali rapor etmelerine rağmen, nesnel testler uykusuzluk çekenler ile iyi uyuyanlar arasında anlamlı bir fark olmadığını göstermektedir. İkinci olarak uykusuzluk çekenler deneyimledikleri stres belirtilerini uyku problemlerini tetikleyen bir etmen olarak görmek yerine uykusuzluğun bir sonucu olarak görmektedirler. Stresörler kaygıya, kaygı da aşırı uyarılmaya sebep olmaktadır. Bu sebeple uykusuzluğu subkortikal yapıların ve amigdala devrelerinin

aşırı uyarılması olarak tanımlayabilmek mümkündür (Carr ve ark., 2003; Desseilles ve ark., 2008; McNamara ve Auerbach, 2001). Üçüncü olarak günlük işlevselliğin bazı alanlarında bozulmalar yaşamalarına rağmen uykusuzluk çekenler, pek çok önemli bilişsel işlevlerini korumaktadırlar. Tüm bunlar noktalar özetlenirse, uykusuzluk çeken kişilerin hayatta kalım ya da üreme ile ilgili tehditlere bağlı olarak görece iyi işlevselliğe sahip ancak uykuya yönelik homeostatik güdülerine direnen kişiler olduğu iddia edilebilir.

Evrimsel bir açıdan bakıldığında aşırı uyarılma ve teyakkuzda olma ile karakterize olan akut uykusuzluğun gerçek ve algılanan stresörlere karşı adaptif bir tepki olduğu iddia edilebilir (McNamara ve Auerbach, 2001; Perogamvros ve ark., 2020). Perogamvros ve arkadaşları (2020) akut uykusuzluğun insanlık tarihinde çoğu zaman gerçek ve güncuel olan ve korku uyandıran tehditlere karşı adaptif bir tepki olduğun ancak günümüzdeki tehditlerin daha çok gelmesi beklenilen tehditler ile bağlantılı ve kaygı olarak deneyimlediğini belirtmişlerdir. Perogamvros ve arkadaşları (2020) göre akut uykusuzluk dönemindeki aşırı uyarılma ve uyanıklık hali klasik koşullanma nedeniyle devam etmekte ve tehditler ortadan kalmasına rağmen korku sönümlenmesinin başarısızlığı sebebiyle uyku problemi kronik uykusuzluğa dönüşebilmektedir. Diğer bir değişle uykusuzluk geçmişteki gerçek ve güncel tehditlere karşı adaptif avantajlar sağlamasına rağmen günümüz dünyasının geleceğe yönelik tehditlerine karşı maladaptif hale gelmektedir. Yakın zamanlı çalışmalar bazı uykusuzluk çekenlerin daha güçlü korku koşullanmasına ve gecikmiş ya da başarısız korku sönümlenmesine sahip olduğunu göstermektedir (Seo ve ark., 2018; Wassing ve ark., 2019). Bu sebeple uykusuzluk çeken kişiler için yatak, yatak odası ya da uyku vakti gibi yansız uyaranların koşullanmamış uyaranlar ile ilişkilendirilmesi sonucu uyanıklık hali oluşabilir. Bu tez çalışması, Perogamvros ve arkadaşlarının (2020) modellerini geliştirerek uyku öncesi zamanda deneyimlenen istenmeyen görsel imgelerin korku koşullanması oluşturabileceğini ve başarısız korku sönümlenmesine sebep olacağını iddia etmektedir.

1.10 Uykusuzluğun Korku Simülasyonu Modeli (UKSM)

Uykusuzluğun Korku Simülasyonu Modeli uyku öncesi dönemde gelecekteki tehditlere yönelik istenmeyen görsel imgelerin bu beklenen tehditlere karşı korku tepkisini simüle ettiğini ve bu simülasyonun korku koşullanmasına sebep olduğunu belirtmektedir. Akut uykusuzluk durumunda uyarılma oluşturacak ve normal uykusuzluğu bozacak iki durum ortaya çıkmaktadır. Bunlardan ilki bir yırtıcıya ya da sosyal düşmanlığa maruz kalmaktır (Perogamvros ve ark., 2020). İkincisi ise gelecekte gerçekleşme ihtimali olan tehditlere yönelik istenmeyen görsel imgeler deneyimlemektir. Bazı bireyler uyku vaktinde istenmeyen görsel imgeler vasıtasıyla gelecek ya da geçmişteki tehdit edici durumları gerçekçi ve canlı bir şekilde simüle ederek korku uyarılması ve koşullanmasına maruz kalmakta olabilirler (Dadds ve ark., 1997; Ji ve ark., 2016; Mertens ve ark., 2020). Modelin özeti Figür 1'de gözükmektedir.



Figür 1 Uykusuzluğun Korku Simülasyonu Modeli

1.11 Görsel İmgeleme ve Görsel İmgeleme Yeteneği

Görsel imgeleme herhangi dışsal bir uyaranın olmadığı durumda (Ji ve ark., 2016), "zihnin gözüyle görmek" (Kosslyn ve ark., 2001, p. 635) şeklinde tanımlanabilecek zihinsel deneyimleri tanımlamaktadır. Görsel imgeleme yeteneği ise kişinin görsel zihin imgeleri oluşturabilmesi anlamına gelmektedir (Hall ve ark., 1985). Görsel imgeleme yeteneğinin üç farklı özelliği bulunmaktadır. Bunlar berraklık, kontrol edebilirlik ve tercih etme'dir. Geçmişte yapılan çalışmalar berraklık, kontrol edebilirlik ve tercih etme gibi görsel imgeleme özelliklerine yönelik kişisel farklılıklar olduğunu ortaya çıkarmıştır. Kadınların erkeklere kıyasla daha berrak görsel imgelere sahip olduğu (Campos ve Pérez, 1988; Campos ve Sueiro, 1993; Wassell ve ark., 2015; White ve ark., 1977), erkeklerin ise imgeleri manipüle etmeyi ölçen performans testlerinden kadınlara kıyasla daha yüksek skorlar aldıkları bulunmuştur (Campos, 2014; Campos ve ark., 2004). Tercih edilme açısından bakıldığında ise güzel sanatlar ve psikoloji öğrencilerinin nesne imgelemesini, mühendislik öğrencilerinin ise uzamsal imgeleme bilişsel stillerini daha çok kullandıkları bulunmuştur (Perez-Fabello ve ark., 2018).

1.12 Görsel İmgeleme ve Görsel Algıyla İlişkisi

Geçmiş çalışmalarda görsel imgelemenin görsel algılamayı taklit ettiği, hatta onun zayıf bir formu olduğu belirtilmiştir (Pearson ve ark., 2015). Gerçekleştirilen sinirbilim çalışmalarında, görsel algılama ve görsel imgelemenin benzer beyin bölgelerini aktive ettiği bulunmuştur. Kosslyn ve arkadaşları (1997) 21 beyin bölgesinden 14'ünün ortak olarak kullanıldığını, Ganis ve arkadaşları (2004) görsel imgeleme için kullanılan beyin bölgelerinin görsel algılamada kullanılan bölgelerin bir alt seti olduğunu, Albers ve arkadaşları (2013) ise görsel imgelemede ortaya çıkan aktivite örtülerinin görsel algıda oluşanlara çok benzediğini belirtmişlerdir.

1.13 Görsel İmgelemenin Etkileri ve Görsel İmgeleme ile Açığa Çıkan Korku Koşullanması

Son 40 yılda yapılan çalışmalar üç tane önemli bulguyu ortaya koymaktadır. Bunlardan ilki bir uyaranı görsel imgeleme ile işlemlemenin psikofizyolojik

uyarılmaya sebep olduğudur (Ji ve ark., 2016; Lang, 1979). İkincisi bir uyaranın görsel imgesinin ortaya çıkardığı fizyolojik etkilerin, uyaranın sözel olarak işlemlenmesine göre daha güçlü olduğudur (Holmes ve ark., 2008; Holmes ve Mathews, 2005; Vrana ve ark., 1986). Üçüncüsü ise daha yüksek berraklığa sahip imgelerin daha yüksek uyarılma ile ilişkili olduğudur (Cui ve ark., 2007; Ji ve ark., 2016; McNeil ve ark., 1993; Rauch ve ark., 2004). Bu bulguların ötesinde yapılan çalışmalar görsel imgelerin korku koşullanmasında uyaranların gerçek dünyada deneyimlenmesine benzer etkiler gösterebildiğini açığa çıkarmıştır (Dadds ve ark., 1997; Mertens ve ark., 2020).

1.14 İstenmeyen Düşünceler

Görsel zihinsel imgeler bizim onları isteyerek oluşturma yeteneğimizden bağımsız olarak istenmeyen ve kontrol edilemeyen bir şekilde de var olabilirler. İstenmeyen düşünceler tekrar eden, kabul edilemez olarak deneyimlenen ve arzulanmayan düşünceler, imgeler ve dürtüler olarak tanımlanmaktadır (Clark ve Purdon, 1995; Rachman, 1978). Bu düşüncelerin içerikleri günahkarca, ahlaksız ve kaygı dolu olabilmektedir (Parkinson ve Rachman, 1981). Yapılan bir çalışmada klinik olmayan örneklemin %79.84'ün istenmeyen düşüncelere sahip olduğu bulunmuştur (Rachman ve de Silva, 1978). İstenmeyen düşüncelerin fiziksel tepkileri nasıl etkilediğini araştıran çalışmalar, istenmeyen düşüncelerin deri iletkenliği, kalp atış hızı ve değişkenliği, kortisol seviyeleri ve merkezi sinir sistemi uyarılması ile olumlu yönde ilişkilere sahip olduğunu göstermektedir (Aldao ve ark., 2013; Hofmann ve ark., 2005; Linz ve ark., 2018; MacNeil ve ark., 2017; Ottaviani ve ark., 2016; Pillai ve Drake, 2015; Rydstedt ve ark., 2011; Weise ve ark., 2013).

İstenmeyen düşüncelerin obsesif kompulsif bozukluk, depresyon (Moritz ve ark., 2019; Wenzlaff, 2002), genel kaygı bozukluğu (Gross ve Eifert, 1990), hipokondria (Arnáez ve ark., 2021; Romero-Sanchiz ve ark., 2017) ve travma sonrası stres bozukluğu (Falsetti ve ark., 2002; Hagenaars ve ark., 2010; Nixon ve ark., 2021) gibi rahatsızlıklarda mevcut olduğu bilinmektedir. İstenmeyen düşüncelerin ve imgelerin bulunduğu diğer bir rahatsızlığın da uykusuzluk olduğu görülmektedir (Clark, 2004;

Nelson ve Harvey, 2002, 2003a, 2003b). Uykusuzluk çeken kişilerin uykuya başlama zamanındaki istenmeyen düşünceleri onların uykuya dalmalarını engelleyebilmektedir (Baglioni ve ark., 2010; Pillai ve Drake, 2015). Geçmiş çalışmalar istenmeyen düşünceler ve uyku öncesi bilişsel aktivitelerinin daha kısa uyku süresi (Kelly, 2002; Nota ve Coles, 2015), uzamış uykuya dalma süresi (Nicassio ve ark., 1985; Nota ve Coles, 2015; Wicklow ve Espie, 2000) ve düşük uyku kalitesi (Baker ve ark., 2015; Hall ve ark., 2000) ile ilişkili olduğunu göstermektedir.

1.15 Uyku Öncesi Uyarılma

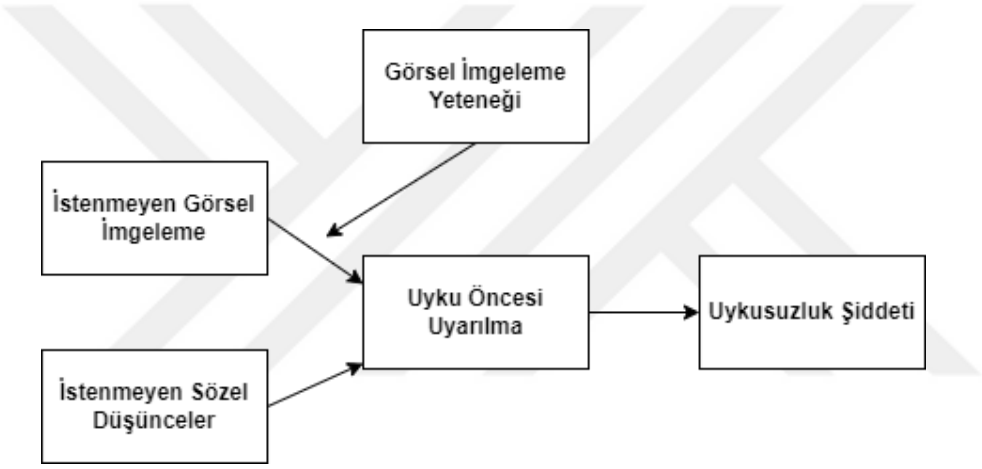
Pek çok uykusuzluk modelinin belirttiği gibi fizyolojik ve bilişsel aşırı uyarılma hali uykusuzluk hastalığına yatkınlığı arttırmakta ve hastalığın devam etmesine katkı sağlamaktadır Marques ve ark., 2015; Nicassio ve ark., 1985; Perlis ve ark., 2016; Riemann ve ark., 2010). Uykusuzluk araştırmaları bağlamında uyku öncesi uyarılma bedensel ve bilişsel uyarılmaya karşılık gelmektedir (Broman ve Hetta, 1994b; Nicassio ve ark., 1985; Riemann ve ark., 2010; Vargas ve ark., 2020). Çalışmalar uykusuzluk çekenlerin iyi uyuyanlara kıyasla daha yüksek uyku öncesi uyarılma puanlarına sahip olduğunu ve yüksek uyku öncesi uyarılma ile uzamış uykuya dalma süresi, kısalmış toplam uyku süresi, azalmış uyku kalitesi, uykuda uyanma sıklığında artış, daha sık sabah erken uyanmaları, daha yüksek uykusuzluk şiddeti arasında ilişki olduğunu göstermektedir (Broman ve Hetta, 1994b; Jansson-Fröjmark ve Norell-Clarke, 2012; Marques ve ark., 2015; Nicassio ve ark., 1985; Palagini ve ark., 2016, 2017; Puzino, Amatrudo, ve ark., 2019; Ruivo Marques ve ark., 2018; Yeh ve ark., 2015).

1.16 Mevcut Çalışma

Mevcut çalışmanın amacı DSM-5 uykusuzluk bozukluğu tanı kriterlerini sağlayan kişileri içeren bir örneklemde istenmeyen görsel imgeler ve sözel düşüncelerin uyku öncesi uyarılma ve uykusuzluk şiddeti üzerindeki etkilerinin incelenmesi ve imgeleme yeteneğinin istenmeyen görsel imgeler ve uyku öncesi uyarılma arasındaki ilişkideki düzenleyici işlevini incelemektir. Bu çalışma ile kronik uykusuzluğun

korku koşullanması hipotezinin direkt olarak test edilmesi değil bu hipotez için öncü kanıtlara ulaşılmaya çalışılmıştır.

Mevcut çalışmanın modeli Figür 2'de görülebilir. Çalışmanın ilk bölümünde Uyku Öncesi Uyarılma Ölçeği ve İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri Türkçeye adapte edilmiştir. Çalışmanın ikinci kısmında ise istenmeyen görsel imgeler, istenmeyen sözel düşünceler, imgeleme yeteneği, uyku öncesi uyarılma ve uykusuzluk şiddeti değişkenlerini içeren bir model yol analizi yöntemi ile test edilmiştir.



Figür 2 Mevcut Çalışmanın Modeli

Mevcut çalışmanın hipotezleri aşağıda görülebilir:

1) İstenmeyen görsel imgeleme, uyku öncesi uyarılmayı anlamlı bir şekilde yordayacaktır.
2) İstenmeyen sözel düşünceler uyku öncesi uyarılmayı anlamlı bir şekilde yordayacaktır.
3) İstenmeyen görsel imgelemeden uyku öncesi uyarılmaya giden yol katsayısı istenmeyen sözel düşüncelerden uyku öncesi uyarılmaya giden yol katsayısından daha büyük olacaktır.

4) Uyku öncesi uyarılma uykusuzluk şiddetini anlamlı bir şekilde yordayacaktır.
5) Görsel imgeleme yeteneği istenmeyen görsel imgeleme ve uyku öncesi uyarılma arasındaki ilişkiyi düzenleyecektir.

BÖLÜM 2

ÇALIŞMA 1: UYKU ÖNCESİ UYARILMA ÖLÇEĞİ VE İSTENMEYEN GÖRSEL İMGELEME VE SÖZEL DÜŞÜNCELER ANKETLERİ'NİN TÜRKÇE KONUŞAN ÖRNEKLEMLERDEKİ PSİKOMETRİK ÖZELLİKLERİ

YÖNTEM

2.1.1 Katılımcılar

Çalışmaya sosyal medya ve ODTÜ Psikoloji Bölümü'nün çevrimiçi veri toplama sistemi, SONA, üzerinden 651 kişi katılmıştır. Açımlayıcı ve doğrulayıcı faktör analizleri veri seti seçkisiz şekilde ikiye bölünerek farklı örneklemler üzerinde gerçekleştirilmiştir. Yakınsak, ıraksak, artımlı ve ayırıcı geçerlilikler tüm ölçekleri dolduran 556 kişinin verisi ile üç haftalık test-tekrar test analizi ise 88 kişilik ayrı bir örneklem üzerinde test edilmiştir. Katılımcıların demografik özelliklerine Tablo 1'ten ulaşabillir.

Tablo 1 Örneklemlerin demografik özellikleri

	İlk Örneklem		İkinci Örneklem		Alt Örneklem		Üçüncü Örneklem	
	Ort.	*SS*	*Ort.*	*SS*	*Ort.*	*SS*	*Ort.*	*SS*
Yaş	28.56	14.11	27.63	13.89	29.25	14.81	22.19	4.98
	Ort.	%	*Ort.*	%	*Ort.*	%	*Ort.*	%
Cinsiyet								
Kadın	207	63.50	218	67.08	349	62.77	71	80.68
Erkek	119	35.89	107	32.92	207	37.23	17	19.32
Eğitim Durumu								
Lise	9	2.76	6	1.85	15	2.70	0	0
Üniversite	290	88.96	287	88.31	482	86.69	100	100
Lisansüstü	27	8.28	32	9.85	59	10.61	0	0
İş Durumu								

Tablo 1 (devamı)

Çalışmıyor	265	81.28	264	81.23	434	78.06	100	100
Çalışıyor	61	18.71	61	18.77	122	21.94	0	0
Medeni Durum								
Bekar	253	77.61	263	80.92	421	75.72	83	94.31
Evli	69	21.17	57	17.54	126	22.66	5	5.68
Boşanmış	4	1.23	5	1.54	9	1.62	0	0
Sosyo-ekonomik Seviye								
Çok düşük	20	6.14	15	4.62	31	5.58	1	1.14
Düşük	47	14.42	47	14.46	80	14.39	10	11.36
Orta	219	67.18	216	66.46	370	66.55	57	64.77
Yüksek	39	11.96	46	14.15	73	13.13	9	21.59
Çok yüksek	1	.31	1	.31	2	.36	1	1.14

2.1.2 Araçlar

İlk çalışmanın verileri Demografik Bilgi Formu, Uyku Öncesi Uyarılma Ölçeği, İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri, Beck Anksiyete Ölçeği, Ruminatif Düşünce Biçimi Ölçeği, Uykusuzluk Şiddeti İndeksi, Nesnesel-Uzamsal ve Sözel Bilişsel Stil Ölçeği, Depresyon-Anksiyete-Stres Ölçeği ve Toplumsal Cinsiyet Rolleri Ölçeği kullanılarak elde edilmiştir.

2.1.3 İşlem

İlk olarak adaptasyonu yapılacak ölçeklerin geliştiricilerine e-posta yolu ile ulaşılarak kendilerinden onam alınmıştır. Daha sonra Orta Doğu Teknik Üniversitesi İnsan Araştırmaları Etik Kurulu'ndan ilk çalışma için etik izin alınmıştır. İngilizce ölçekler ilk olarak iki araştırmacı tarafından Türkçeye çevrilmiş ve daha sonra iki dilli diğer bir araştırmacı tarafından bu çeviriler birleştirilmiştir. Birleştirilen formların anlaşılırlığı 25 kişinin katıldığı bir pilot çalışma ile test edilmiş. Sonuçlar ve katılımcıların önerileri doğrultusunda formlara son halleri verilmiştir. Çalışmada kullanılan tüm araçlar katılımcılara rastgele bir sırada çevrimiçi şekilde sunulmuştur.

2.1.4 İstatistiksel Analiz

Verilerin analizi SPSS 25 ve JASP 0.16.1 programları kullanılarak gerçekleştirilmiştir. Açımlayıcı ve doğrulayıcı faktör analizleri alanyazındaki kriter

ve önerilere göre gerçekleştirilmiştir. Örneklem sayılarının belirlenmesinde gene alanyazındaki önerilere başvurulmuştur. Yakınsak-ıraksal, artımlı ve ayırıcı geçerlik sırasıyla Pearson korelasyon katsayıları, çoklu regresyon analizi ve bağımsız t-test ile alt örneklem üzerinde test edilmiştir.

2.2. BULGULAR

2.2.1 Uyku Öncesi Uyarılma Ölçeği'nin Psikometrik Özellikleri

2.2.1.1 Açımlayıcı Faktör Analizi

Uç değerlere sahip katılımcıların çıkarılması ile 322 kişiden elde edilen veriler ile değerlendirilmiştir. Sonuçlar ölçeğin iki faktörlü (bilişsel ve bedensel uyku öncesi uyarılma) bir yapıya sahip olduğunu göstermektedir. Ölçeğin bilişsel uyku öncesi uyarılma alt ölçeğine ait olan 8. maddesi yeterli faktör yüküne sahip olmadığı için ölçekten çıkarılmıştır.

2.2.1.2 Doğrulayıcı Faktör Analizi

Uç değerlere sahip katılımcıların çıkarılması ile 314 kişiden elde edilen veriler ile değerlendirilmiştir. Açımlayıcı faktör analizi sonucunda ortaya çıkan 15 maddeli ve iki faktörlü yapı test edilmiştir. Madde 3-4, Madde 6-7 ve Madde 9-11'in hataları arasına koyulan hata kovaryansları sonucu modelin geçerli bir uyuma sahip olmuştur.

2.2.1.3 Yakınsak ve Iraksak Geçerlilik

Uç değerlere sahip katılımcıların olmaması sebebiyle 556 kişinin verileri kullanılmıştır. Sonuçlar Uyku Öncesi Uyarılma Ölçeği'nin bilişsel uyarılma alt ölçeği ile Ruminatif Düşünce Biçimi Ölçeği (r = .72, p < .001), Uyku Öncesi Uyarılma Ölçeği'nin bedensel uyarılma alt ölçeği ile Beck Anksiyete Envanteri'nin bedensel alt ölçeği (r = .67, p < .001), Uyku Öncesi Uyarılma Ölçeği ile Uykusuzluk Şiddeti İndeksi (r = .65, p < .001) arasında pozitif ilişkiler olduğunu göstermektedir. Bunlara ek olarak Toplumsal Cinsiyet Rolleri Ölçeği ile Uyku Öncesi Uyarılma Ölçeği (r = .25, p < .001), bilişsel uyarılma alt ölçeği (r = .28, p < .001) ve bedensel uyarılma alt

ölçeği (r = .15, p < .05) arasında pozitif ilişkiler olduğu bulunmuştur. Sonuçlara göre Uyku Öncesi Uyarılma Ölçeği'nin yakınsak ve ıraksak geçerliğini desteklemektedir.

2.2.1.4 Artımlı Geçerlilik Geçerlik

Uç değerlere sahip katılımcıların çıkarılması ile 555 kişiden elde edilen veriler ile değerlendirilmiştir. Uykusuzluk şiddetinin yordanan değişken olduğu iki basamaklı regresyon analizinin ilk basamağında modele depresyon, anksiyete ve stres değişkenleri girilmiştir. İlk model %25'lik bir varyansı açıklamıştır. Daha sonra bilişsel ve bedensel uyku öncesi uyarılma değişkenlerinin modele eklenmesi sonucu açıklanan varyans %42.60'ye çıkmış ve modelde sadece bilişsel ve bedensel uyku öncesi uyarılma değişkenlerinin uykusuzluk şiddetini anlamlı olarak yorduyor olduğu bulunmuştur. Bu sonuçlar Uyku Öncesi Uyarılma Ölçeği'nin artımlı geçerliliğini desteklemektedir.

2.2.1.5 Ayrıcı Geçerlilik

Uç değerlere sahip katılımcıların olmaması sebebiyle 556 kişinin verileri kullanılmıştır. Uykusuzluk çeken kişiler Uykusuzluk Şiddet İndeksi'nden 8 puan ve üstü alan kişiler olarak belirlenmiştir (Bastien ve ark., 2001). Gerçekleştirilen bağımsız t-testleri uykusuzluk çekenlerin normal uyuyan kişilere kıyasla anlamlı olarak daha yüksek bilişsel ve bedensel uyku öncesi uyarılma puanlarına sahip olduğunu göstermiştir. Bu sonuçlar Uyku Öncesi Uyarılma Ölçeği'nin ayırıcı geçerliliğini desteklemektedir.

2.2.1.6 Güvenirlik

556 katılımcının verileri kullanılarak gerçekleştirilen iç tutarlılık analizi Uyku Öncesi Uyarılma Ölçeği, bilişsel uyarılma alt ölçeği ve bedensel uyarılma alt ölçeğinin sırası ile .92, .93 ve .86 Cronbach'ın alfa değerlerine sahip olduğu göstermiştir. 88 katılımcının olduğu üçüncü örneklemde gerçekleştirilen test-tekrar test analizi, Uyku Öncesi Uyarılma Ölçeği, bilişsel uyarılma alt ölçeği ve bedensel uyarılma alt ölçeğinin sırası ile .90, .90, .83 sınıf-içi korelasyon katsayılarına sahip olduğunu açığa

çıkarmıştır. Sonuçlar Uyku Öncesi Uyarılma Ölçeği'nin Türkçe formunun iyi güvenirliğe sahip olduğunu göstermektedir.

2.2.2 İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri'nin Psikometrik Özellikleri

2.2.2.1 Açımlayıcı Faktör Analizi

Uç değerlere sahip katılımcıların çıkarılması ile 317 kişiden elde edilen veriler ile değerlendirilmiştir. Sonuçlar ölçeğin iki faktör yapısına sahip olduğunu göstermektedir. Ölçek 10 maddeli istenmeyen görsel imgeleme ve 10 maddeli istenmeyen sözel düşünceler olmak üzere 2 faktörden oluşmaktadır.

2.2.2.2 Doğrulayıcı Faktör Analizi

Uç değerlere sahip katılımcıların çıkarılması ile 313 kişiden elde edilen veriler ile değerlendirilmiştir. Açımlayıcı faktör analizi sonucunda ortaya çıkan 20 maddeli ve iki faktörlü yapı test edilmiştir. Madde 1-2, Madde 2-3, Madde 6-10, Madde 12-13, Madde 15-16, Madde 16-20'nin hataları arasına koyulan hata kovaryansları sonucu, modelin geçerli bir uyuma sahip olduğu görülmüştür.

2.2.2.3 Yakınsak ve Iraksak Geçerlilik

Uç değerlere sahip katılımcıların olmaması sebebiyle 554 kişinin verileri kullanılmıştır. Sonuçlar İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri'nin istenmeyen görsel imgeleme (r = .37, p < .001) ve sözel düşünceler (r = .17, p < .001) alt ölçekleri ile Nesnesel-Uzamsal ve Sözel Bilişsel Stil Ölçeği'nin nesne imgesel alt ölçeği arasında pozitif ilişkiler olduğunu göstermiştir. Ayrıca İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri ve alt ölçeklerinin Depresyon, Anksiyete ve Stres Ölçeği'nin alt ölçekleri ile anlamlı ilişkilere sahip olduğu bulunmuştur. Son olarak Toplumsal Cinsiyet Rolleri Ölçeği ile görsel imgeleme alt ölçeği (r = .21, p < .001) ve sözel düşünceler alt ölçeği (r = .22, p < .001) arasında pozitif ilişkiler tespit edilmiştir. Sonuçlara göre İstenmeyen Görsel

İmgeler ve Sözel Düşünceler Anketleri'nin yakınsak ve ıraksak geçerliğini desteklemektedir.

2.2.1.6 Güvenirlik

556 katılımcının verileri kullanılarak gerçekleştirilen iç tutarlılık analizi İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri, görsel imgeleme alt ölçeği ve sözel düşünceler alt ölçeğinin sırası ile .96, .93 ve .94 Cronbach'ın alfa değerlerine sahip olduğu göstermiştir. 88 katılımcının olduğu üçüncü örneklemde gerçekleştirilen test-tekrar test analizi, İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri, görsel imgeleme alt ölçeği ve sözel düşünceler alt ölçeğinin sırası ile .91, .83, .91 sınıf-içi korelasyon katsayılarına sahip olduğunu açığa çıkarmıştır. Sonuçlar İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri'nin Türkçe formunun iyi güvenirliğe sahip olduğunu göstermektedir.

BÖLÜM 3

ÇALIŞMA 2: MODEL TESTİ

YÖNTEM

3.1.1 Katılımcılar

Çalışmaya sosyal medya ve ODTÜ Psikoloji Bölümü'nün çevrimiçi veri toplama sistemi üzerinden 1444 kişi katılmıştır. Bu katılımcılardan 168 tanesinin DSM-5'in Uykusuzluk Bozukluğu tanı kriterlerini sağladığı ve uykusuzluk şiddeti skorlarının 8'den büyük olduğu bulunmuş ve model testi bu 168 katılımcının verileri ile gerçekleştirilmiştir. Katılımcıların demografik özelliklerine ve uyku ile ilgili betimleyici bilgilere Tablo 3'ten ulaşılabilir.

Tablo 3 Demografikler ve Örneklemin Uyku Betimleyici Bilgileri

	Ort.	*SS*
Yaş	25.55	5.26
Uykusuzluk belirtilerinin sıklığı (haftada kaç gün)	5.16	1.52
Uykusuzluk belirtilerinin süresi (yıl)	4.11	3.54
	N	%
Cinsiyet		
Kadın	126	75.00
Erkek	42	25.00
Eğitim		
Üniversite	84	50.00
Lisansüstü	84	50.00
İş Durumu		
Çalışmıyor	109	64.88
Çalışıyor	59	35.12
Medeni Durum		
Bekar	147	87.50
Evli	19	11.31
Boşanmış	2	1.19

Tablo 3 (devamı)

Sosyo-ekonomik Seviye		
Çok düşük	32	19.05
Düşük	46	27.38
Orta	80	47.62
Yüksek	9	5.35
Çok yüksek	1	.60
Uykusuzluk Belirtileri		
Uykuya Dalma Güçlüğü	150	89.29
Uykuyu Sürdürme Güçlüğü	45	73.21
Çok Erken Uyanma	36	21.43

3.1.2 Araçlar

İkinci çalışmanın verileri Demografik Bilgi Formu, Uyku Öncesi Uyarılma Ölçeği, İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri, Uykusuzluk Şiddeti İndeksi, Nesnesel-Uzamsal ve Sözel Bilişsel Stil Ölçeği – Nesnesel İmgeleme Alt Ölçeği ve RBTSE-5 Uykusuzluk Bozukluğu tanı kriterlerini içeren sorular kullanılarak elde edilmiştir.

3.1.3 İşlem

İlk olarak Orta Doğu Teknik Üniversitesi İnsan Araştırmaları Etik Kurulu'ndan ikinci çalışma için etik izin alınmıştır. Çalışmada kullanılan tüm araçlar katılımcılara rastgele bir sırada çevrimiçi şekilde sunulmuştur.

3.1.4 İstatistiksel Analiz

Verilerin analizi SPSS 25 ve JASP 0.16.1 programları kullanılarak gerçekleştirilmiştir. Yol analizi alanyazındaki kriter ve önerilere göre gerçekleştirilmiştir. Örneklem sayısının belirlenmesinde gene alanyazındaki önerilere başvurulmuştur.

3.2. BULGULAR

3.2.1 Modeldeki Değişkenlere Ait Betimleyici İstatistikler

Modelde kullanılmış değişkenlerin betimleyici istatistiklerine ve Pearson korelasyonu katsayılarına Tablo 4'ten ulaşılabilir. Uç değerlerin çıkarılması sonucu 166 katılımcının verileri kullanılmıştır.

Tablo 4 Modeldeki değişkenlere ait betimleyici istatistikler ve korelasyonlar

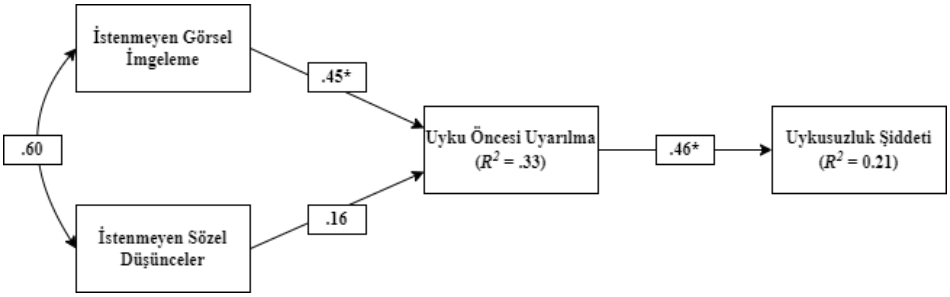
Ölçekler	1	2	3	4	*Ort.*	*SS*	*Min*	*Maks*
1. İstenmeyen Görsel İmgeleme	-				33.30	9.92	10.00	50.00
2. İstenmeyen Sözel Düşünceler	.69***	-			36.96	8.87	10.00	50.00
3. Görsel İmgeleme Yeteneği	.37***	.31***	-		51.60	12.52	19.00	75.00
4. Uyku Öncesi Uyarılma	.56***	.47***	.25**	-	45.27	10.34	18.00	70.00
5. Uykusuzluk Şiddeti	.35***	.28***	.16*	.46***	15.36	4.31	8.00	26.00

Not. * $p < .05$, ** $p < .01$, *** $p < .001$.

3.2.2 Yol Analizi

İstenmeyen görsel imgeler, istenmeyen sözel düşünceler, uyku öncesi uyarılma ve uykusuzluk şiddetini içeren bir yol modeli kurulmuştur. Modelin iyi uyuma sahip olduğu bulunmuştur ($\chi^2(2) = 2.76$, $p = .25$, SRMR = .03, RMSEA = 0.05, *90% CI* = [.00,.17], TLI =0.98, CFI = 0.99). Modeldeki direkt ilişkiler istenmeyen görsel imgelerin ($\beta = .45$, $p < .001$) ve istenmeyen sözel düşüncelerin ($\beta = .16$, $p = .07$) uyku öncesi uyarılmayı anlamlı bir şekilde yordadığını ve uyku öncesi uyarılmanın ($\beta = .46$, $p < .001$) da uykusuzluk şiddetini anlamlı bir şekilde yordadığını göstermiştir. Dahası direkt olmayan etkilerin analizi, istenmeyen görsel düşüncelerin (IE = .20, $p < .001$) uyku öncesi uyarılma üzerinden uykusuzluk şiddetini anlamlı olarak

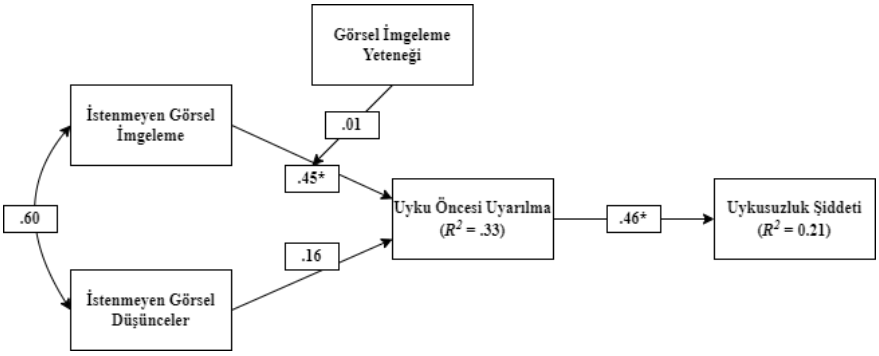
etkilediğini ancak istenmeyen sözel düşüncelerin (IE = .07, p = .08) uyku öncesi uyarılma üzerinden giden etkisinin uykusuzluk şiddetini anlamlı olarak etkilemediğini göstermiştir. Model Figür 3'te görülebilir. Sonuçlar hipotez 1, 3, ve 4'ü desteklemektedir.



Not 1. * p < .001
Not 2. Değişkenler arasındaki standart yol katsayıları gösterilmiştir. .10 = düşük etki, .30 = orta etki ve .50 = yüksek etki (Cohen, 1992).
Figür 3 Yol Modeli

3.2.3 Düzenleyici Analizi

Hipotez 5'i test etmek için istenmeyen görsel imgeler, görsel imgeleme yeteneği, istenmeyen görsel imgeler ve görsel imgeleme yeteneğinin etkileşimi ve uyku öncesi uyarılma değişkenlerinin olduğu bir model kurulmuştur. Sonuçlar görsel imgeleme yeteneğinin istenmeyen görsel imgeler ve uyku öncesi uyarılma arasındaki ilişkide düzenleyici etkisi olmadığını göstermiştir, R^2 = .01, F (1, 162) = 2.56, p = .11. Bu sebeple hipotez 5 desteklenememiştir.



Not 1. * p < .001
Not 2. Değişkenler arasındaki standart yol katsayıları gösterilmiştir. .10 = düşük etki, .30 = orta etki ve .50 = yüksek etki (Cohen, 1992).

Figür 4 Düzenleyici Değişken ile Beraber Yol Modeli

BÖLÜM 4

TARTIŞMA

4.1 Uyku Öncesi Uyarılma Ölçeği'nin Psikometrik Özellikleri

Açımlayıcı faktör analizi sonuçları Uyku Öncesi Uyarılma Ölçeği'nin geliştirildiği çalışma (Nicassio ve ark., 1985), Janssons-Fröjmark ve Norell-Clarke'ın çalışması (2012), Almanca (Gieselman ve ark., 2012), Urduca (Namra ve Tazvin, 2014), Portekizce (Ruivo Marques ve ark., 2018) ve Japonca (Okajima ve ark., 2020) adaptasyon çalışmaları ile uyumlu olarak iki faktörlü bir yapı açığa çıkarmıştır. Doğrulayıcı faktör analizi sonuçları benzer şekilde ikili faktör yapısını doğrulamıştır. Yakınsak, ıraksak, artırımlı ve ayırıcı geçerlik analizleri Uyku Öncesi Uyarılma Ölçeği'nin geçerliğini desteklemektedir. Benzer şekilde güvenirlik analizlerinin sonuçları da Uyku Öncesi Uyarılma Ölçeği'nin güvenirliğini destekleyici sonuçlar ortaya koymuştur.

4.2 İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri'nin Psikometrik Özellikleri

Açımlayıcı faktör analizi sonuçları İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri'nin geliştirildiği çalışma (McCarthy-Jones ve ark., 2012) ile uyumlu olarak iki faktörlü bir yapıya sahip olduğunu göstermektedir. Doğrulayıcı faktör analizi sonuçları benzer şekilde ikili faktör yapısını doğrulamıştır. Yakınsak ve ıraksak geçerlik analizleri İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri'nin geçerliğini desteklemektedir. Benzer şekilde güvenirlik analizlerinin sonuçları da İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri'nin güvenirliğini destekleyici sonuçlar ortaya koymuştur.

4.3 Çalışma 1'in Kısıtlılıkları ve Gelecek Çalışmalar İçin Öneriler

Çalışma 1'in ilk kısıtlılığı katılımcıların ağırlıklı olarak kadın olmasıdır. Sonraki çalışmaların cinsiyet açısından daha dengeli bir örneklem kullanmaları önerilebilir. Benzer şekilde katılımcıların çoğunun üniversite öğrencisi olması sonuçların genelleştirilebilmesini zorlaştırmaktadır. Sonraki çalışmaların bu anlamda da daha yüksek temsiliyete sahip örneklemleri kullanmalıdır. Son olarak doğrulayıcı faktör analizinde bazı maddelerin hataları arasına kovaryasyon koyulması modele, modelin içindeki değişkenler ve onların arasındaki ilişkiler ile açıklanamayan eklemeler yapıldığı anlamına gelmektedir (Hermida, 2015).

4.4 Çalışma 1'in Çıkarımları

Uyku Öncesi Uyarılma Ölçeği'nin Türkçe formu deneysel ve gözlemsel uyku tıbbı çalışmalarında kullanılabilir. Benzer şekilde Uyku Öncesi Uyarılma Ölçeği'nin skorları kronik uykusuzluk çeken kişileri belirlemekte kullanılabilir. Ayrıca Uyku Öncesi Uyarılma Ölçeği'nin skorları uykusuzluk ve uyku problemlerini azaltmayı hedefleyen müdahaleleri içeren çalışmalarda sonuç değişkeni olarak kullanılabilir.

İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri görsel ve sözel süreçlerin etkilerinin arasındaki farkları değerlendirmek için kullanılabilir. Benzer şekilde İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri, istenmeyen düşüncelerin psikopatolojilerdeki rolünü inceleyen çalışmalarda kullanılabilir. Son olarak İstenmeyen Görsel İmgeler ve Sözel Düşünceler Anketleri istenmeyen görsel imgeleme ve sözel düşünceleri hedefleyen müdahalelerde sonuç ölçümü olarak kullanılabilir.

4.5 Hipotez 1, 2 ve 3'e ait Bulgular

Yol analizinin sonuçları sadece istenmeyen görsel imgelemenin uyku öncesi uyarılmayı anlamlı bir şekilde yordadığını göstermiştir. Aynı zamanda istenmeyen görsel imgelemenin etkisinin istenmeyen sözel düşüncelerden daha güçlü olduğu görülmüştür. İstenmeyen düşünceler uyku öncesi uyarılmaya ait varyansın %32.70'ni açıklamıştır. Bu sonuçlar hipotez 1 ve 3'ün desteklendiği göstermektedir. Geçmişte

yapılan çalışmalar da benzer şekilde görsel imgelemenin sözel işlemlemeye göre daha güçlü fizyolojik ve duygusal uyarılma yarattığını göstermektedir. (Cuthbert ve ark., 2003; Holmes ve ark., 2008; Holmes ve Mathews, 2005; Lang, 1979; Vrana ve ark., 1986).

4.6 Hipotez 4'e ait Bulgular

Yol analizinin sonuçları uyku öncesi uyarılmanın uykusuzluk şiddetini anlamlı bir şekilde yordayabildiğini göstermektedir. Uyku öncesi uyarılmanın uykusuzluk şiddetine ait varyansın %21.20'sini açıklayabildiği görülmektedir. Bu sonuç geçmişteki çalışmaların bulguları ile uyumludur (Buysse ve ark., 2011; Harvey, 2002; Lundh ve Broman, 2000; Morin, 1993; Perogamvros ve ark., 2020; Spielman ve ark., 1987).

4.7 Hipotez 5'e ait Bulgular

Analizler görsel imgeleme yeteneğinin istenmeyen görsel imgeleme ile uyku öncesi uyarılma arasındaki ilişkide düzenleyici rolünün olmadığını göstermiştir. Bunun sebeplerinden bir tanesi çalışmada kullanılan görsel imgeleme yeteneğinin imgelemenin berraklığı değişkenini kapsayan daha büyük bir yapı olmasıdır. Görsel imgeleme yeteneğinin toplam puanı imgelemenin berraklığını, düzenleyici anlamda temsil edememiş olabilir. Diğer bir olasılık düzenleyici etkinin çalışmadaki katılımcıların aldığı en düşük görsel imgeleme yeteneği puanı olan 19'un altında bir değerde anlamlı olduğudur.

4.8 Çalışma 2'nin Kısıtlılıkları ve Gelecek Çalışmalar için Öneriler

Çalışma 2'nin birinci kısıtlılığı yol analizinin aslında değişkenler arasındaki nedenselliği açıklamaktan ziyade değişkenleri arasında nedensel ilişkiler olan bir modeli yanlışlayabileceğidir (Streiner, 2005). Bu sebeple mevcut bulguları inceleyecek deneysel ve uzamsal çalışmalar yapılması gerekmektedir. İkinci olarak uykusuzluk bozukluğu olan katılımcılar bir psikiyatrist görüşmesi yerine anket ve ölçekler kullanılarak belirlenmeye çalışılmıştır. Üçüncü olarak örneklemin büyük çoğunluğu genç üniversite öğrencisi kadınlardan oluşmaktadır. Sonraki çalışmaların

temsil gücü daha yüksek örneklemler ile yürütülmesi daha iyi olacaktır. Son olarak çalışmada kullanılan istenmeyen düşünceler ölçekleri, bu özelliği genel bir kişilik özelliği olarak ölçmektedir. Alanyazında uyku öncesi istenmeyen görsel imgeleme ve sözel düşünceleri değerlendiren bir ölçüm aracı olmadığı için gelecekti çalışmalar bu ölçekleri geliştirmeye odaklanabilir.

4.9 Çalışma 2'nin Klinik Çıkarımları

Çalışma 2'ya ait birkaç klinik çıkarımda bulunulabilir. Sonuçlar istenmeyen görsel imgelerin uyku öncesi uyarılma üzerinden uykusuzluk şiddeti üzerindeki etkisinin anlamlı olduğunu göstermiştir. Bu durumda istenmeyen görsel imgeleri ve bir ihtimal istenmeyen sözel düşünceleri azaltan müdahalelerin uyku problemlerini azaltacağı söylenebilir. Harvey (2005) bu amaçla kullanılabilecek üç müdahale stratejisinden bahsetmektedir. Bunlardan ilki istenmeyen düşünceleri kontrol etmeye yönelik çabalardan kurtulmaktır. İstenmeyen düşünceleri bastırmaya yönelik çabaların bu düşüncelerin daha güçlü bir şekilde geri dönmesine sebep olduğu bilinmektedir (Wegner, 1994). Bu yüzden mindfulness meditasyonu istenmeyen düşünceleri kontrol etme çabalarını ortadan kaldırmak için kullanılabilir (Espie ve Ellis, 2016). İkinci bir yöntem olarak istenmeyen düşüncelerden kaçmak yerine onlara yaklaşmaktır (Harvey, 2005). Bu amaçla dışavurumcu yazım kullanılabilir (Lepore, 1997). Üçüncü strateji istenmeyen düşünceler üzerindeki dikkatimizi başka bir yöne çekmektir (Harvey, 2005). Bu amaçla rahatlama ve dikkati başka yöne odaklayan görsel imgeleme egzersizleri kullanılabilir.

Alanyazında Tetris oynamanın görsel kısa süreli belleği meşgul ederek istenmeyen görsel imgelemeyi engellediği ve şiddetini düşürdüğünü gösteren çalışmalar vardır (Horsch ve ark., 2017; Iyadurai ve ark., 2020; Kanstrup, Kontio, ve ark., 2021; Kanstrup, Singh, ve ark., 2021; Kessler ve ark., 2018, 2020; Skorka-Brown ve ark., 2014, 2015). Bu sebeple mavi ışık yaymayan bir ekrandan uyku öncesi zamanda oynanan Tetris istenmeyen görsel imgelemenin azaltılmasında kullanılabilir. Buna ek olarak alanyazındaki bazı müdahaleler uyku problemlerini temsil eden görsel

imgelerin tanımlanması ve olumlu bir hale dönüştürülmesine odaklanmıştır (Schmid ve Steil, 2019).

Mevcut çalışmanın sonuçları ve Uykusuzluğun Korku Simülasyonu Modeli gözden geçirildiğinde görsel imgelemeyi kullanan özgün bir müdahale yöntemi önerilebilir. Bu yöntem Simüle Edilmiş Korku Sönmesi Müdahalesi olarak isimlendirilmektedir. Uykusuzluk çeken kişiler yatmadan önce kendilerini her zaman uyudukları uyku ortamının içinde imgeleyemeye başlayarak normal bir şekilde uykuya daldıklarını, uykularını sürdürdüklerini ve sabah uyandıklarını imgelerler. Bu imgeleme egzersizi görsel kısa süreli hafızayı kullandığı için istenmeyen görsel imgelerin ortaya çıkmasını engellerken, korku koşullanması ile ilişkili bağlamsal ve işaretli uyaranların tekrardan uykuya dalma ile eşleştirilmesine yardımcı olmaktadır.

4.10 Sonuç

Sonuç olarak istenmeyen görsel imgelemenin uyku öncesi uyarılma ve uykusuzluk şiddeti üzerindeki etkilerinin anlaşılmasının ve çalışılmasının uykusuzluk bozukluğuna yönelik tedavilerin geliştirilmesinde önemli bir rol oynayabileceği görülmektedir. Simüle Edilmiş Korku Sönmesi Müdahalesi tek başına bir müdahale olarak kullanılabileceği gibi Uykusuzluk için Bilişsel Davranışçı Terapi'ye uykusuzluğun altındaki klasik koşullanmayı ortadan kaldıran bir strateji olarak entegre edilebilir. Bu yönde gelecekte çalışmalar yapılması önerilmektedir.

APPENDIX R: CURRICULUM VITAE

PERSONAL INFORMATION

Surname, Name:

Nationality:

Date and Place of Birth:

Marital Status:

Phone:

Email:

EDUCATION

Degree	Institution	Year of Graduation

WORK EXPERIENCE

Year	Place	Enrollment

FOREIGN LANGUAGES

Advanced English, Basic German.

PUBLICATIONS

1) Türkarslan, K. K. (2022). The Replication Crisis and Psychotherapy Efficacy Studies. Studies in Psychology, 42(1), 1-41.
2) Okay, D., Türkarslan, K. K., Çevrim, M., & Bozo, Ö. (2022). Sleep Go Away, I Don't Have Much Time Left to Live: Is Bedtime Procrastination Related to Perceived Time Left in Life?. Journal of Turkish Sleep Medicine, 9(1), 57-63.
3) Türkarslan, K. K. (2021). Children's affectionate and assertive attitudes towards their parents: the Oedipus complex or parent-offspring conflict? Integrative Psychological and Behavioral Science. (Early Access).
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8) Türkarslan, K. K., Okay, D., Çevrim, M., & Bozo, Ö. (2020). Life is short, stay awake: Death anxiety and bedtime procrastination. The Journal of General Psychology, 147(1), 43-61.

BOOK CHAPTERS

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APPENDIX S: TEZ İZİN FORMU / THESIS PERMISSION FORM

ENSTİTÜ / INSTITUTE

Fen Bilimleri Enstitüsü / Graduate School of Natural and Applied Sciences ☐

Sosyal Bilimler Enstitüsü / Graduate School of Social Sciences ☒

Uygulamalı Matematik Enstitüsü / Graduate School of Applied Mathematics ☐

Enformatik Enstitüsü / Graduate School of Informatics ☐

Deniz Bilimleri Enstitüsü / Graduate School of Marine Sciences ☐

YAZARIN / AUTHOR

Soyadı / Surname : Türkarslan
Adı / Name : Kutlu Kağan
Bölümü / Department : Psikoloji / Psychology

TEZİN ADI / TITLE OF THE THESIS (**İngilizce** / English): The Roles of Intrusive Visual Imagery and Visual Imagery Ability in Insomnia Disorder

TEZİN TÜRÜ / DEGREE**:** **Yüksek Lisans** / Master ☐ **Doktora** / PhD ☒

Tezin tamamı dünya çapında erişime açılacaktır. / Release the entire work immediately for access worldwide. ☒

Tez iki yıl süreyle erişime kapalı olacaktır. / Secure the entire work for patent and/or proprietary purposes for a period of **two years. *** ☐

Tez altı ay süreyle erişime kapalı olacaktır. / Secure the entire work for period of **six months**. ***** ☐

Yazarın imzası / Signature ..... **Tarih** / Date .....